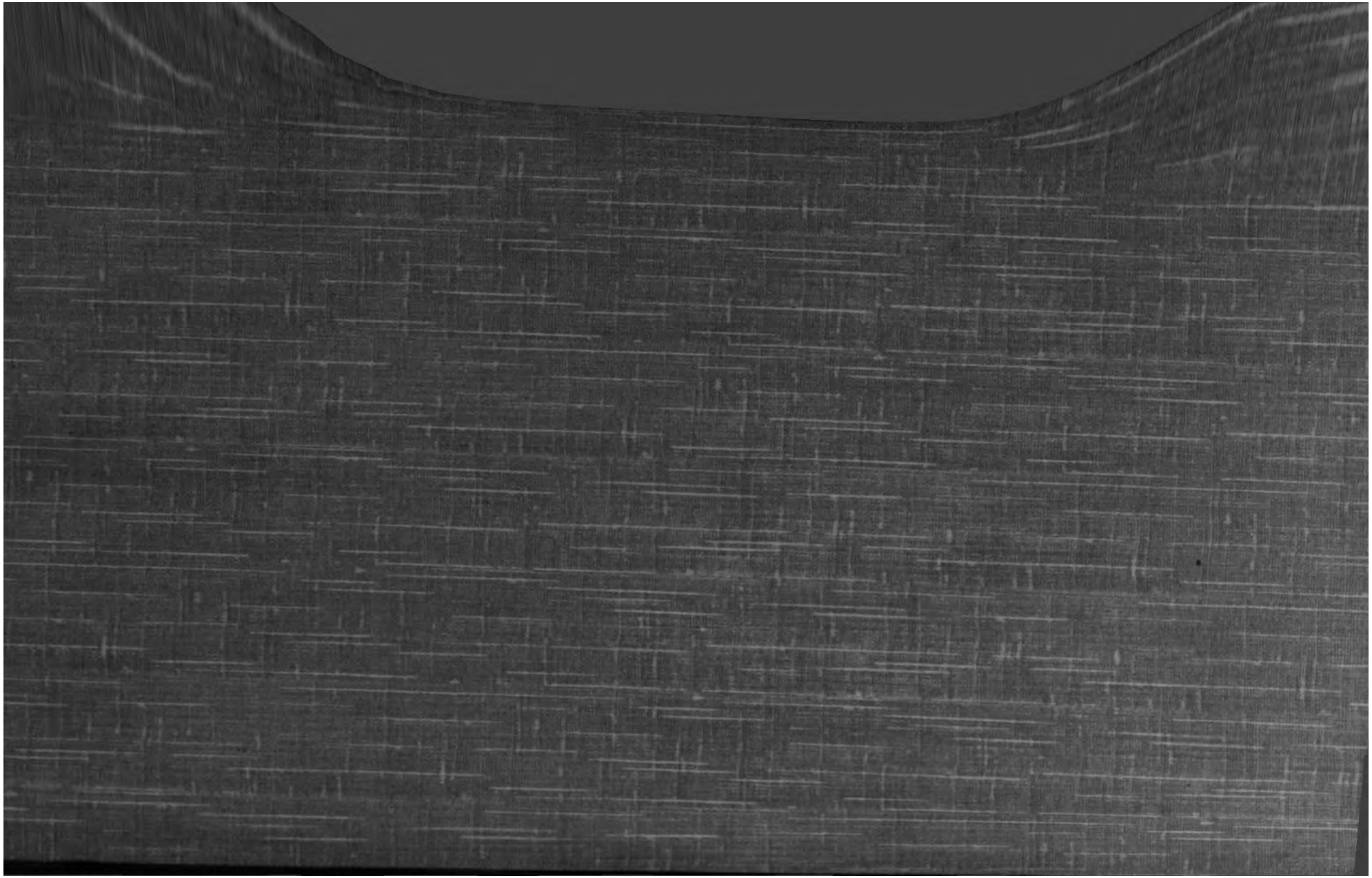

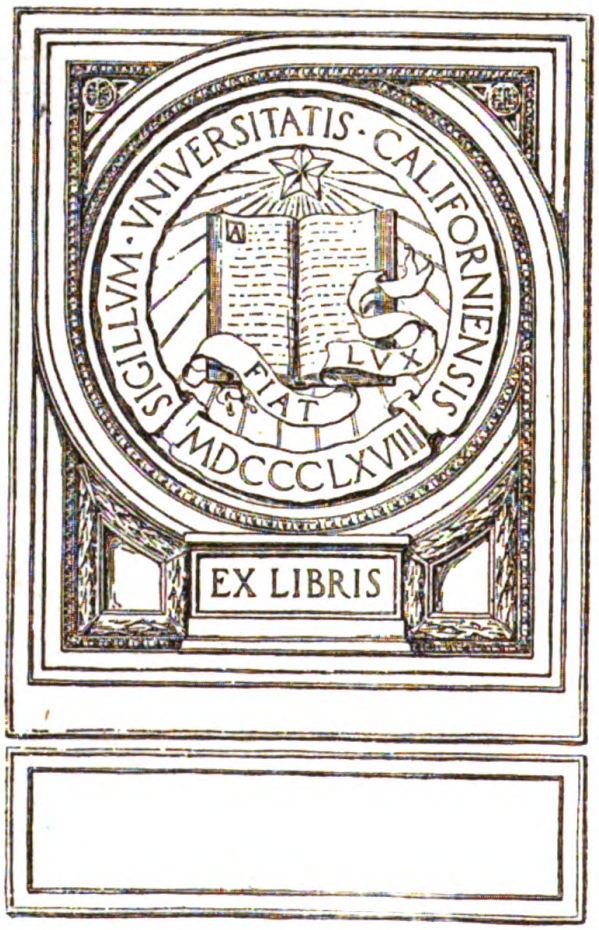
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REVIEW

OF

NEUROLOGY AND PSYCHIATRY

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Review
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Original Articles

A PLEA FOR THE STUDY OF THE INTERMEDIO-LATERAL CELL-SYSTEM OF THE SPINAL CORD.[1]

By ALEXANDER BRUCE, M.A., M.D.,
Physician to the Edinburgh Royal Infirmary ;

and

J. H. HARVEY PIRIE, B.Sc., M.B.,
Clinical Tutor in the Medical Wards, Edinburgh Royal Infirmary.

OUR main object in bringing this communication before the Society is to draw attention to the importance of the study of a system of nerve cells in the spinal cord which, in our opinion, appears to deserve more attention than it has yet generally received. We are all more or less familiar with the large motor cells in the anterior horns of the cord: their distribution is well known, as are also many of the pathological changes which they undergo, and considerable progress has been made in the establishment of their relation to the innervation of individual muscles. In like manner with regard to the cells of the vesicular column of Clarke, much is already known. Its distinctness as a group, and the characteristic form of its cells, so unlike anything else in the cord, has led to recognition of its exact position and distribution in the cord, and recent methods of research appear to have proved beyond doubt that its axones

[1] A paper read before the Medico-Chirurgical Society of Edinburgh on November 7, 1906.

are continued into the direct cerebellar tract of Flechsig. In addition, however, to these two outstanding cell-systems, there remain at least two other which have not received the same amount of study. There are large numbers of small polygonal cells scattered irregularly at the base of the anterior horns, whose function and distribution still remain to be determined; and, secondly, there is the cell-system which forms the subject of our communication to-night.

This cell-system was first observed by Lockhart Clarke in 1851. In 1859, in a second paper read before the Royal Society of London, he described more fully the distribution and character of the cells, and gave it the name "intermedio-lateral tract," which it now generally bears. This term, as used by Clarke, indicates that it is a tract, column, or system of nerve cells situated along the lateral margin of that portion of the grey matter which is intermediate between the anterior and posterior cornua. Clarke stated also that the tract extended from the lower end of the cervical to the upper end of the lumbar enlargement, and that it was not found in these enlargements, but reappeared above and below them.

For a long time after the publication of Clarke's papers, anatomists seem to have in the main contented themselves with merely noting its existence, and tended to locate it in the apex of the lateral horn of the cord. It was only gradually that it became recognised that the lateral horn in which the tract was located was not the same as the lateral projection of the anterior horns in the lumbar and cervical enlargements, and that the cells of the intermedio-lateral tract were of different form and character from those of the anterior cornua. It was not until Waldeyer published in 1888 his classical paper on the spinal cord of the gorilla that any material advance in our knowledge was made. Waldeyer showed that the tract was situated not merely in the lateral horn of the grey matter, but that it extended backwards as far as the angle between the lateral and posterior horns, to a point in immediate relation to the formatio reticularis. While agreeing with Clarke that the cells of that part of the tract which was situated in the lateral horns are not found in the cervical and lumbar enlargements, he traced those cells overlying the formatio reticularis through both enlargements, thus making the intermedio-lateral cell-system co-extensive with the spinal cord.

In 1905 one of us (A. B.) submitted the intermedio-lateral tract to an exhaustive examination by means of serial sections, and arrived at the following, amongst other conclusions :—

(*a*) In agreement with Waldeyer, that the intermedio-lateral tract was composed of cells in and near the tip of the lateral horn (which may be termed *apical* cells), and also of cells at the angle of the formatio reticularis (the *reticular* cells).

(*b*) That the upper limit of the tract was in the middle of the eighth cervical segment, and the lower limit at the lower end of the second, or possibly at the upper end of the third lumbar segment.

(*c*) That the cells were arranged in peculiar groups or masses, sometimes quite separated from each other and sometimes quite continuous.

(*d*) That these groups were apparently characteristic for each segment, and were on the whole fairly symmetrical on the two sides.

(*e*) That the number of the cells was immensely greater than has been hitherto realised.

The following are the total numbers counted in each segment :—

	Left.	Right.
C. 8	595	429
D. 1	1867	1720
D. 2	3931	3635
D. 3	7297	7471
D. 4	3623	3803
D. 5	7958	7407
D. 6	8105	8030
D. 7	8739	8282
D. 8	6223	5943
D. 9	9011	8987
D. 10	8903	10,203
D. 11	6261	6498
D. 12	7888	8545
L. 1	7053	6765
L. 2	1123	1464
Total	88,577	89,182

Other points were established, but for these the original paper in the *Transactions of the Royal Society of Edinburgh*, Vol. xlv., Part 1, 1906, may be consulted.

It cannot be said that the possible function of the intermedio-lateral cell-system was seriously considered until Gaskell published his epoch-making paper on the sympathetic system in the *Journal of Physiology* in 1886. In this paper he showed that while *all* the roots of the spinal cord were connected with ganglia of the sympathetic system by means of fine non-medullated fibres (grey rami communicantes), only a limited number of these roots were also connected with sympathetic ganglia by fine medullated fibres (white rami communicantes). That limited number extended from the second (afterwards corrected to the first) thoracic or dorsal to the third or fourth lumbar in the cat. Besides these there were two additional white rami from the lower sacral cord. Gaskell was struck by the fact that the longitudinal extent of origin of these white rami corresponded with that of the vesicular column of Clarke and suggested that this column was probably the spinal centre for these fibres. Subsequently he stated that the intermedio-lateral tract also acted as a centre of origin. It will be noted that the longitudinal distribution of the cells of the intermedio-lateral tract corresponds exactly with that of the origin of the white rami. The mere existence of such a coincidence is not in itself sufficient, however, to establish a connection between the cells of the intermedio-lateral tract and the white rami communicantes. The most satisfactory way to prove this would no doubt be to trace the fine fibres directly from the cells to the anterior roots from which the white rami arise. That still remains to be done. The problem has, however, been attacked in another way, namely, by studying the changes which take place in the cells of the cord after division of nerve roots emerging from it. It is now established that under certain circumstances, by some retrograde influence, the cells connected with the divided nerve fibres undergo a peculiar and easily detectable change, viz. a displacement of the nucleus towards one surface, and a disappearance of the chromatic granules. To this process the term *réaction à distance* has been applied. It may be regarded as demonstrated that if, after the section of a bundle of nerve fibres, certain nerve cells degenerate in the

way mentioned, these cells are connected with the divided fibres. By these means it has been found by Anderson that, on dividing the cervical sympathetic, the cells of the intermedio-lateral tract in the upper region underwent the *réaction à distance*. His results have been completely confirmed by Herring, who found a degeneration of the cells in the tract beginning above at the level of the superficial origin of the eighth cervical root, being most marked at the level of the first and second dorsal, and diminishing downwards as far as the sixth dorsal segment. Both these observers found that the cell-change did not extend beyond the intermedio-lateral tract.

Onuf and Collins, however, have given a more extensive origin to the sympathetic fibres, tracing it not only into the lateral columns, but into certain paracentral cells, into cells in the posterior cornua, etc., while Biedl has actually found after section of the splanchnics a degeneration in the lateral horns of the lowest cervical, and in the anterior horns of the upper dorsal extending as high as the sixth cervical and as low as the fifth dorsal nerve. Other observers have found an even more extensive degeneration, but their results are of such a nature as to suggest some flaw in the operative or histological technique. It appears to us on reviewing the literature, that it is justifiable to assume that the origin of the cervical cord is as Anderson and Herring have stated it, and that the analogy of the relationship of the splanchnics to the cord suggests a similar origin. Of this, however, an absolutely complete experimental proof still remains to be made.

Gaskell arrived at the conclusion that the white rami were efferent fibres, and that of those some terminated in vertebral ganglia, others passed through these and terminated in prevertebral ganglia, which lay in more or less immediate relation to viscera. He made the bold generalisation that those which terminated in the vertebral ganglia were vaso-motor, while those which terminated in the distal or prevertebral ganglia were all vaso- or viscero-inhibitory. This view has not been upheld, and we owe to Langley, Anderson, Schäfer, Rose Bradford, Bayliss and Starling, François Franck, and other workers too numerous to mention here, the establishment by the sounder method of experiment of the functions of the various roots. If, as seems justifiable, we may infer that the effects produced

by stimulation of the roots represent some, if not all, of the effects resulting from the normal action of the cells of origin of these roots, then we obtain by these experimental methods data as to the functions of the different parts of the intermedio-lateral tract.

The accompanying table shows the effects of stimulating the various roots of the sympathetic systems.

	1	2	3	4	5	6	7	8	9	10	11	12	13	1	2	3
Dilatation of Pupil (1)	**1**	**2**	3													
Retraction of Nictating Membrane: Separation of Eyelids; Protrusion of Eye (1)	1	**2**	**3**	4	(5)											
Blood-Vessels of Head (1)	(1)	2	**3**	**4**	5											
Secretion of Submaxillary Gland in Cat (1)	(1)	**2**	3	4	5											
Acceleration of Heart (1)	1	**2**	**3**	**4**	5	(6)										
Hairs of Face and Neck (1)			(3)	**4**	**5**	**6**	7									
Vaso-Motor Fibres to, and Sweat Glands of, Fore-Foot (1)				4	5	6	**7**	8	9	(10)						
Spleen (2)			(3)	(4)	5	**6**	**7**	**8**	9	10	(11)	(12)	(13)	(1)		
Vaso-Constrictors of Portal System (3)			(3)	4	**5**	**6**	**7**	**8**	**9**	10	(11)					
Viscero-Motor and Viscero-Inhibitory to Small Intestine (4)						6	7	**8**	**9**	**10**	**11**	**12**	**13**	**1**	2	(3) **(4)**
Inhibitory to Small Intestine (5)						6	7	8	**9**	**10**	**11**	**12**	13	1		
Vaso-Motor to Liver, Pancreas, Small Intestine; 1st Part of Large Intestine (6)					(5)	6	7	8	9	10	11	**12**	**13**	1	2	
Vaso-Motor to Kidney (7)				4	5	6	7	8	9	10	**11**	**12**	**13**	(1)	(2)	(3) **(4)**
Vaso-Motor and Sweat Secretion of Hind-Foot (1)											11	12	13	**1**	**2**	**3**

The numbers within brackets indicate levels where the resulting stimulation was faint, inconstant, or doubtful.
The numbers in heavy type indicate levels at which stimulation gave the maximum result.

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[1] Langley, "Schäfer's Physiology," Vol. ii., p. 616, *et seq.*, with further references to literature.

[2] Schäfer and Moore. *Virchows Arch.*, 1877, p. 181.

[3] Bayliss and Starling. *Journ. Physiol.*, Vol. xvii., p. 120.

[4] Bunch. *Journ. Physiol.*, Vol. xxii., p. 357.

[5] Bechterew and Mislawsky. *Arch. de Physiol. norm. et path.*, 1889, p. 243.

[6] François Franck and Hallion, *Ibid.*, 1896 and 1897.

[7] Bradford. *Journ. Physiol.*, 1896, p. 358.

All the sympathetic fibres for the upper part of the body, with the exception of those for the acceleration of the heart and for the sweat secretion and circulation of the fore-foot, pass to the superior cervical ganglion. In the two exceptions mentioned the fibres terminate in the ganglion stellatum. Those fibres which innervate the abdominal viscera pass into the solar ganglia. The fibres for the circulation and sweat secretion of the hind foot enter the sixth and seventh lumbar and the first and second sacral ganglia.

These anatomical and physiological details would only have an academic value for us, and would not be in place at a meeting of our Society unless they could be linked up to clinical and pathological facts. We are in a position to bring before the Society notes of two cases in which the symptoms indicated an involvement of the sympathetic system, and in which we have found lesions of the intermedio-lateral tract at levels suggested by the results of physiological experiment—lesions which therefore appear to us to be adequate to have produced the symptoms in question.

In one case loss of sweating in the lower extremities had developed during the course of an acute atrophic paralysis in an adult. The other case was one of acute exophthalmic goitre, in which death took place during an access of hyper-thyroidism.

Case I. X. Y., unmarried, aged 30, had been in somewhat imperfect general health for a considerable number of months, owing to a succession of attacks of influenza and to overwork. For some three months previous to the acute onset of his illness he had complained of pains about the middle of the dorsal region, and in the shoulders and feet. There had also been several crops of purpuric spots on the feet and legs. These, as well as the pains referred to, had been regarded as probably of rheumatic origin. On one occasion some difficulty of micturition had been noted, and some ten days or so previous to the sudden onset of the paralysis this difficulty recurred. There also appeared a slight tendency to stumble while walking, some unsteadiness in standing, and a degree of tingling and numbness in both lower extremities, particularly in the right. These symptoms were attributed simply to weakness, and, although the patient always felt very tired, he continued to take a certain amount of exercise. On the 1st of September he noted that he walked with difficulty, and felt much exhausted after a short game of golf. On the night of the 1st September 1905 he slept soundly, and on waking found that he was completely paralysed, both as to motion and sensation, from the waist downwards.

On examination, on the 4th September, there was complete flaccid paralysis of the muscles of the abdomen and of both lower extremities.

There was almost complete loss of all forms of sensibility below the level of the 5th intercostal space, although there were still some slight traces of tactile sensibility in the skin of the abdomen. At the 5th intercostal space sensibility was impaired, and there was no hyperæsthetic zone. Above the 5th interspace, sensation and motor power were quite intact. There was no pain or tenderness over any part of the spinal column or elsewhere in trunk or limbs.

The deep reflexes of the lower limbs were entirely absent. On tickling the soles of the feet, there was no response on the left, and a doubtful extensor response on the right side. The cremasteric and abdominal reflexes were present on both sides.

Slight twitching movements of the right leg occurred frequently. There was retention of urine and fæces. The temperature was 101-102° F. The heart, lungs, and urinary system were normal. Apart from the above-mentioned influenzal and rheumatic attacks, patient had suffered from no other diseases of importance, except the ordinary infectious diseases of childhood. The family history was unexceptionable.

Progress of the case as regards :—

1. Motility.

From the date of the first examination till the date of death, on January 10th, 1906, there was never the slightest return of any voluntary motor power in the lower limbs. The muscles remained flaccid throughout and atrophied very rapidly.

The knee-jerks remained constantly absent, and the plantar reflexes were seldom obtainable, although it was thought that there was occasionally an indefinite extensor response on the right side. The anal reflex was marked, and the sphincter ani retained its tone.

The patient suffered much from muscular twitchings and jerkings in both lower extremities, anterior abdominal wall, and muscles of the lower part of the back. These twitchings were sometimes slight, almost tremor-like ; at other times large, strong jerks, moving the limbs and bending the back. They were considerably influenced by posture, occurring most frequently when the patient was lying on his back, but almost invariably ceasing when he was turned on one or the other side.

Passage of a catheter not infrequently set up strong, almost tetanic contraction of the recti abdominis ; frequently also the constrictor urethræ went into spasm, which effectually prevented the passage of the instrument.

2. Sensibility.

Touch.—Sensibility to light touch began to return within a few days of the onset, and returned over the lower part of the trunk, the whole of the left lower limb and most of the right, except the outer side of the leg and part of the inner side about the ankle. There was a spot on the ball of the right great toe where the tactile sensibility was particularly acute. Elsewhere the return of sensibility was not equally perfect, however, and touch was not

always correctly localised. The patient never felt the passage of the catheter, but there was a slight degree of sensibility in the rectum.

Pain Sense.—In the course of a fortnight there was a slight return of sensibility to pain over the lower dorsal areas, particularly on the left side, and in the same region there was a slight appreciation of heat and cold. Below this level there was no return of sensibility to pain or temperature.

Throughout the greater part of the course of the illness there was a marked subjective sensation of cold in the paralysed areas. This might appear spontaneously after perspiration, or after the slight exposure necessary for nursing purposes. It was present even with a considerable degree of fever, when the skin felt hot and dry to the touch. It often resembled what is found during the cold stage of a malarial attack.

3. Trophic Functions.

In addition to the muscular wasting of the lower extremities already noted, there was a marked and rapid wasting of the whole body.

Within a few days of the onset of the disease, several small bullæ formed on the left tibia and on the inner side of the right knee, and a small subcutaneous hæmorrhage where the knee had been allowed to come into contact with the other leg.

On the dorsum of the right foot several erythematous patches appeared, and over the outer malleolus a small bulla like those on the left leg. With care and suitable treatment these all healed.

A fortnight after the onset there appeared over the tip of the sacrum a small sore—not a typical bed-sore, but rather a hæmorrhagic spot, although the skin over it gave way. This healed, but about six weeks from the commencement of the disease, typical bed-sores appeared on the buttocks, and, despite all treatment, gradually increased in size and were with difficulty kept from becoming septic sores.

4. (*a*) Micturition.

At first there was complete retention, but after three weeks the function became of the involuntary automatic type, *i.e.* the urine accumulated in the bladder until a certain pressure was reached, then a contraction set in, the tension of the "internal sphincter" was overcome, and a certain amount of urine was forcibly ejected. The bladder was never completely emptied, however, a certain amount of residual urine always remaining.

Cystitis very rapidly ensued, and the bladder had to be regularly washed out. The capacity of the bladder gradually diminished; 12 to 15 oz. of residual urine were usually present at first; this gradually diminished until at the end only about 1 oz. remained. The introduction of a corresponding amount of boracic lotion always caused a forcible contraction. There was no sensation connected with micturition. The sense of pressure usually associated with a full bladder was also absent.

(*b*) Defæcation.

At first there was complete inaction of the lower bowel; later the action of the bowels could be kept fairly regular with the aid of medicine and of

occasional enemata. Although the movement of the bowels was entirely beyond the control of the will, certain indefinite sensations usually made the patient aware of the approaching passage of fæces, although these were also passed sometimes unconsciously.

5. Temperature and Sweating.

Patient's temperature was of a hectic, swinging type during all four months, at first ranging from 99-101°. After a fortnight it fell almost to normal for a few days, but again began to rise and fall, and gradually the minima rose till the temperature was seldom below 101-102°. On one occasion the temperature rose as high as 106°. The time of occurrence of these elevations of temperature was quite irregular. With many of them there were rigors or shiverings followed by profuse perspirations. The distribution of the sweating was remarkable. Above the level of the sixth intercostal nerve after each attack of fever, the perspiration was very profuse. Below a sinuous line situated about two and a half inches above Poupart's ligament there was absolute loss of sweating, except over a small area on the inner side of both thighs. Between this sinuous line and the level of the fifth dorsal nerve the skin of the abdomen perspired in irregular patches. The area from which perspiration was entirely absent is shown in Fig. 1.

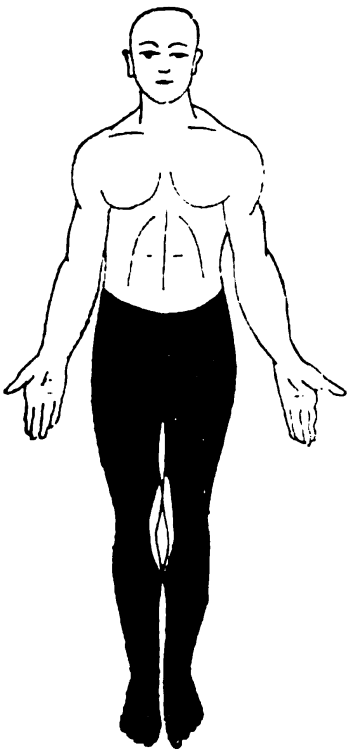

Fig. 1.—The area from which sweating was absent is indicated in black. Note the small unshaded area on the inner side of both thighs.

7. General Condition.

The patient's condition became gradually worse, in spite of all treatment. The heart and lungs were uninvolved. A fair quantity of nourishment was taken, but despite this there was emaciation progressing to an extreme degree. The mental capacities ultimately became gradually dulled, and death ensued from sheer exhaustion, four months after the onset.

Permission was obtained for an examination of the spinal cord.

Examination of the Cord.

Examination of the spinal cord showed that the meninges were throughout its whole length quite normal. The cord was smaller than normal, and the lumbo-sacral enlargement was somewhat soft in consistence. The anterior spinal vein was dilated. The veins of some of the posterior roots in the lower dorsal and lumbo-sacral region were greatly dilated.

On section of the cord, the grey matter in the lumbar and sacral enlargements was so greatly congested as to have an

almost general red colour, on which minute hæmorrhages could be distinctly seen. This congestive and hæmorrhagic condition extended into the lower dorsal region and gradually diminished in an upward direction, ceasing altogether about the level of the sixth or seventh dorsal segment.

Microscopical Examination.

At the levels of the lower dorsal segments and the lumbo-sacral enlargements, partially organised thrombi were found in the anterior spinal vein (which they did not, however, completely occlude), and also in some of the arteries of the posterior roots and in a few of the lateral coronary branches passing towards the intermedio-lateral tract.

The grey matter of the anterior cornua displayed a great congestion of its arterioles and capillaries. A similar congestion could be seen in the base of the posterior cornua, in the lateral cornua, and in the margin of the grey matter between the lateral and posterior horns. In the anterior horn the finest capillaries were all greatly injected and tortuous, and there were very numerous minute hæmorrhages. In a considerable number of instances the extravasated blood had found its way into the lymphatic spaces surrounding the motor nerve cells of the anterior cornua. Red cells were also frequently seen in the perivascular spaces, and also more or less scattered throughout the neuroglia. A careful search throughout the whole of the affected area failed to show any escape of leucocytes or any definite proliferation of the connective tissue cells. The motor cells in the affected area were almost all in a state of degeneration, the degree of which, however, varied within wide limits. A few apparently healthy cells, with well-marked polygonal outline and a centrally-placed nucleus and deeply staining, sharply defined tigroid bodies, were seen scattered irregularly amongst other markedly degenerated cells. The majority of the cells, however, showed displacement of nucleus. In a large number the nucleus was either close to or projected out from the periphery of the cell. There was a marked chromatolysis, the tigroid bodies being replaced by a turbid, finely granular cytoplasm. Within this cytoplasm there were not infrequently vacuoles varying in size. In a great many of the cells there were also masses of golden-yellow granular pigment, in some large

enough to occupy almost the whole cell. The affected cells were swollen, and had largely lost their angular, polygonal outline, their dendrites being markedly retracted. As already stated, there was a marked congestion of the vessels in the lateral cornua, and nearer examination revealed the fact that there were numerous small hæmorrhages amongst the cells of the intermedio-lateral tract, both at the apex of the lateral horn and in the reticular group of cells. There were also on both sides almost symmetrical hæmorrhages in the vessels leading from the surface of the cord to the tip of the lateral horn, and also from others within the substance of the formatio reticularis close to the margin of the grey matter. A few vessels in the substance of the formatio reticularis were found to be completely thrombosed, and one or two minute arterioles contained organised thrombi. Throughout the tenth, eleventh, and twelfth dorsal and the first and second lumbar segments, both the apical and reticular groups on both sides of the intermedio-lateral tract showed degenerative changes analogous to those found in the large motor cells of the anterior cornua, the cells showing marked lateral displacement of nuclei (see Figs. 2 and 3), and disappearance more or less complete of chromatic granules. Some of the cells were also remarkably swollen and their shape distorted.

Similar degenerative changes were found in the small polygonal cells of the anterior cornua (Waldeyer's "middle cells"), and also in the large solitary cells of Schwalbe in the posterior cornua. The cells of Clarke's column appeared unduly swollen, and many showed complete chromatolysis. The known variety of changes in normal cells of this system make it difficult to speak definitely as to the incidence of degeneration. In the lateral columns of the lower dorsal, lumbar and sacral enlargements there was a degree of sclerosis in the pyramidal tract. The direct cerebellar tract was only very slightly affected, a fact which would indicate that the cells of the column of Clarke had escaped degeneration.

In considering this case we have to draw special attention to two outstanding features: the extensive degree of acute atrophic paralysis and its association with vascular paresis in the lower limb, and the loss of sweating in almost the whole of the lower part of the body below a level drawn about two inches above Poupart's ligament. The first explanation that might naturally

suggest itself is that one lesion is responsible for the motor paralysis and for the loss of the function of perspiration; but the association of the two conditions is too rare for that possibility to be entertained. It is remarkable how seldom a persistent dryness of skin is referred to in cases of acute atrophic paralysis due to lesions of the anterior cornual cells, and it is obvious that the muscular paralysis and atrophy must be attributed to the affection of the anterior cornua, and that the residual symptoms of the vaso-motor and secretory disturbances in the skin must be attributable to some additional lesion. That additional lesion is in all probability the hæmorrhage and congestion, and the consequent degeneration of the cells of the intermedio-lateral tract in the tenth, eleventh, and twelfth dorsal and in the first and second lumbar segments. This corresponds fairly closely to the levels where Langley has found that stimulation of the sympathetic roots causes sweating in the hind foot of the dog. It is evident from Higier's and Schlesinger's writings that there is a special area for sweat secretion in the lower limb, corresponding closely to that found in our case, and we would suggest that in similar, or in more limited affections of vaso-motor and secretory disturbances in the lower limb, a special examination of this area of the intermedio-lateral tract should be made.

The second case in which we have found a lesion of the intermedio-lateral tract was one of exophthalmic goitre, in which death took place apparently from hyperthyroidism. The opportunity of examining the spinal cord of this case we owe to the kindness of Dr Gulland, who has supplied the following notes:—

Case II. Mrs M., aged 47. Admitted to Chalmers' Hospital on 22nd May 1906. Died 5th June. Duration of illness, eleven months. Began with an attack of diarrhœa eleven months ago, since when she has been nervous and weak. Swelling of the neck was noticed first in January 1906. Exophthalmos was not noticed by her friends.

Status Præsens.—Marked tremor; slight exophthalmos. Right lobe of thyroid most enlarged. Pulse averages 120 to 130. Presystolic and systolic murmurs. No albumen or sugar. Very nervous and excited.

Course.—Temperature above normal throughout; for the first week ran between 98 and 100; second week, between 99·5 and 100·5, rose to 103·4 just before death. Nothing found in lungs or elsewhere to account for this.

From June 1st she became very noisy and excited; pulse became weaker and more rapid; the heart became dilated. Speech became inarticulate; excreta were passed in bed; urine contained acetone—no sugar. On the 4th she became unconscious, and died at 6.10 on the 15th.

Post-mortem.—Same day. *Thymus* large; *thyroid* large, pale; very little colloid; active proliferation of gland tissue, so that it presented the appearance of a secreting gland; some fibrosis. *Left supra-renal* large and cystic. *Kidneys* showed some chronic interstitial nephritis with an acute toxic condition superadded. *Liver:* chronic venous congestion, with some fatty change and areas of early cirrhosis. *Spleen:* recent infarcts, acute congestion. *Stomach:* congestion and hæmorrhagic infiltration. *Heart:* right auricle dilated, other cavities small, chronic thickening of mitral and aortic valves; no recent endocarditis; some fibrous change in myocardium. *Lung:* no pneumonia; certain amount of œdema and congestion. *Marrow* was a lymphocytic, not a myelocytic marrow. No other abnormalities in the body.

The spinal cord was handed to us by Dr Wade immediately after the post-mortem examination, and placed at once in 10 per cent. formalin solution. On the following day, on dividing the cord into segments, we observed minute hæmorrhages at the outer side of the grey matter on both sides at the lower part of the 2nd, in the 3rd, and in the 4th dorsal segments, and in no others. Microscopic examination demonstrated certain lesions in the grey matter and in the immediately adjacent white matter. These changes were partly in the blood-vessels and partly in the cells of the intermedio-lateral tract. The most obvious morbid appearances were capillary hæmorrhages. These were almost symmetrical as regards position, but were considerably larger on the left side than on the right. They were situated at the apex of the posterior horns, immediately within the outer margin of the grey matter, and extended backwards into the posterior horns and forwards as far as the lateral horn. They were apparently quite recent, probably merely terminal hæmorrhages. Another prominent vascular change was the hyaline degeneration of the tunica media of the arterioles of the lateral columns, which led towards the lateral horns, and also of others in the posterior columns directed towards the root of the posterior horns. The anterior spinal artery and its branches, as well as the coronary branches to the anterior cornua, were quite free from this hyaline change. It was pronounced in the lower part of the 2nd, in the 3rd, 4th, and 5th segments, but it was also found in a gradually decreasing extent in the same group of arterioles as far down as the lower dorsal segments.

The changes in the nerve cells to which we desire to draw special attention, were almost entirely limited to those contained in the intermedio-lateral tract. In the 1st dorsal segment there was a slight chromatolysis in a few of the cells of the anterior

cornua, but with this exception the motor cells remained entirely normal. In marked contrast to these normal motor cells were the cells of the intermedio-lateral tract. These in the 3rd and 4th dorsal segments were, almost without exception, more or less affected; in the 2nd and 5th the involvement was to a less extent, and it was very slight in the 1st and 6th segments. As may be seen in Figs. 4 and 5, the abnormal cells showed somewhat similar degenerations to those described in our previous case. In a large proportion there was a lateral displacement of nucleus, and greater or less disappearance of chromatic granules. In addition, some of the cells were swollen or vacuolated, while many were shrunken and distorted and altogether devoid of nucleus or chromatin. A careful examination of the grey matter showed that the cells of the intermedio-lateral tract had been practically exclusively affected in this special manner.

It is beyond the purpose of this paper to enter into a discussion of the question as to whether the nervous lesion or the affection of the thyroid is the primary one in exophthalmic goitre. We wish in the meantime to content ourselves with drawing the attention of the Society to the fact that the segments involved are those which experiment shows to be the segments which give origin to the roots, stimulation of which produces retraction of the upper eye-lid, protrusion of the eye-ball, acceleration of the heart, and sweating and vaso-regulation of the upper part of the body. We would suggest that future investigations of the sympathetic system should include the cells of the intermedio-lateral tract of the whole thoracic region, as well as the cells of the bulbar autonomic system, which Dana has shown to be involved in one case of exophthalmic goitre.

Onuf and Collins, in their valuable work on the sympathetic nervous system, have also drawn attention to the possible connection of the sympathetic cells in the cord with the diseases which are known to be of sympathetic origin, and with others which are so frequently regarded as being merely due to functional disturbances. Lannois and Porot, in the *Revue de Médecine*, 1903, have published a case of erythromelalgia, with gangrene of the extremities, associated with extensive involvement of the intermedio-lateral tract. With that exception comparatively little has apparently been done to establish a relation of lesions of the tract with morbid phenomena. A possible explanation

of this may lie in the fact that changes in the nerve cells may be in many instances not obviously destructive, but of a nature which can only be demonstrated by modern methods of histological investigation. It is easy to understand how fine changes in cells of a system whose function was not even suspected had escaped detection. If we succeed in drawing the attention of the Society to the importance of the intermedio-lateral tract, the purpose of our paper will have been fulfilled.

Description of Figures.

Figs. 2 and 3 represent the appearances presented by the degenerated cells of both intermedio-lateral tracts in the lower dorsal and upper lumbar region in the case of acute atrophic paralysis.

Fig. 2.—In the centre of the field is a swollen cell with a partial chromatolysis and a displaced nucleus.

Fig. 3 shows, above and to the left, two cells with partial chromatolysis. In the cell to the left the nucleus is displaced upwards and to the left. In that to the right the nucleus is situated to the right of the cell. Two smaller cells above the two just described show similar displacement of nucleus. On the right side of the section and near the centre there are other two cells in both of which displacement of nucleus is detectable. About one centimetre above the large capillary in the lower part of the field is seen a large cell in which the half to the left was pale from the presence of a yellow granular pigment. In the right half there was marked chromatolysis.

Figs. 4 and 5 represent the degeneration of cells found in the intermedio-lateral tracts, especially in the second, third, fourth, and fifth dorsal segments, in the case of exophthalmic goitre.

Fig. 4.—Middle of second dorsal segment, right half. To the right of the centre is a swollen cell showing a granular cell-substance. To the left of this is another cell with displacement of nucleus, loss of chromatin and a distinct vacuole. In the lower part of the field are other cells with displaced nuclei.

Fig. 5.—Lower end of second dorsal segment, left side. Almost in the middle of the field is a swollen cell with loss of nucleus and chromatin granules. To the left, above, and to the right of this cell are three cells showing distinct displacement of nucleus. That situated above the large granular cell also shows small vacuoles. One cell near the top of the right half of the field shows also a displacement of nucleus.

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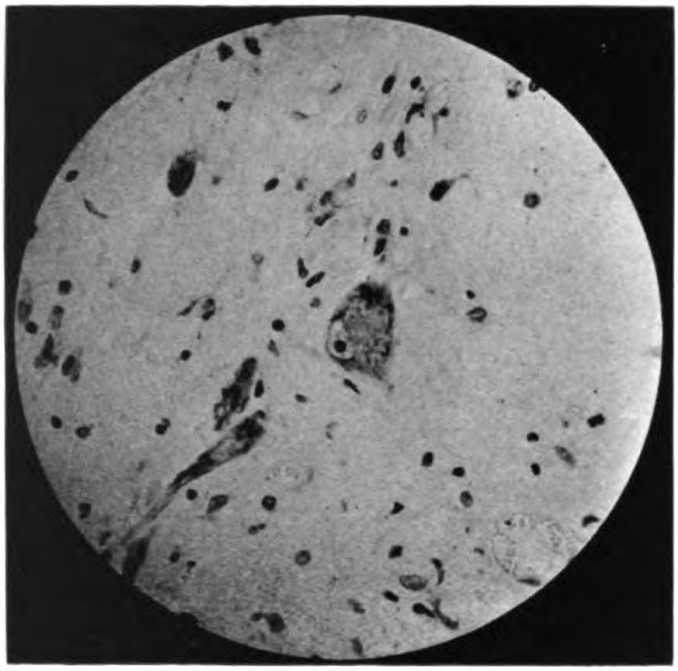

FIG. 2.

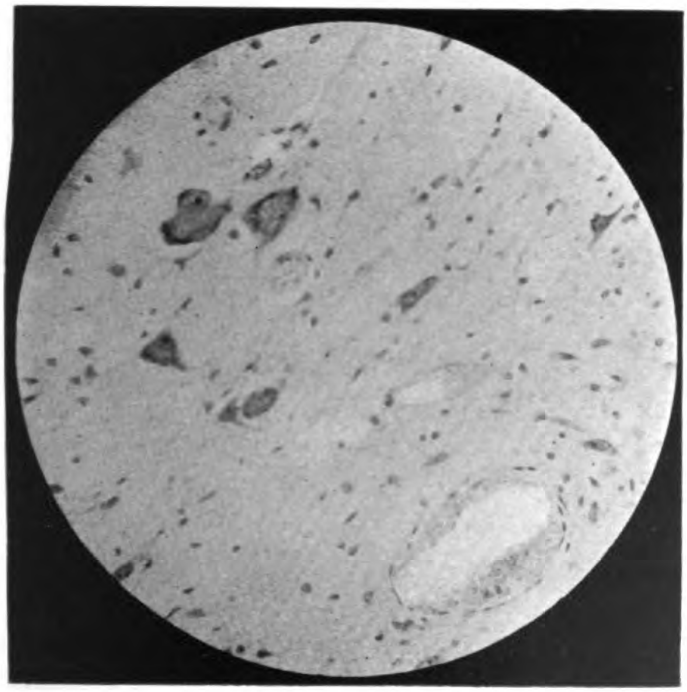

FIG. 3.

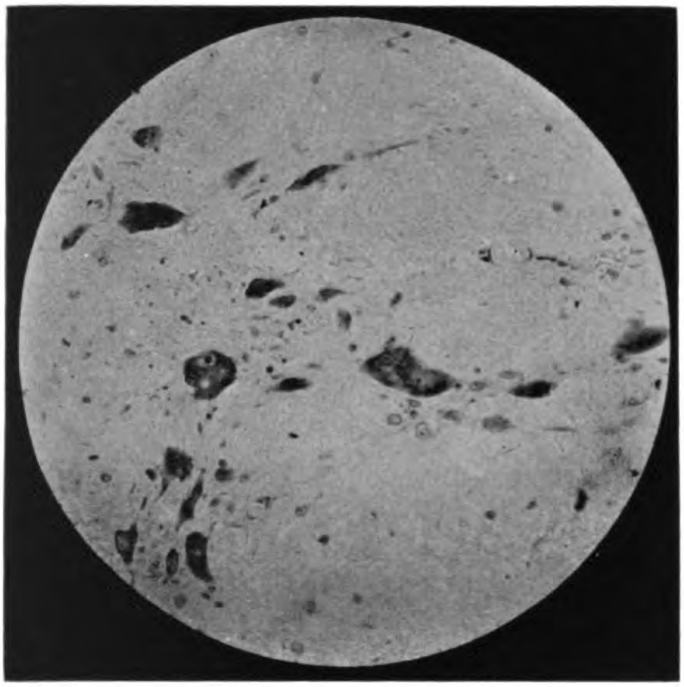

FIG. 4.

FIG. 5.

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A REPORT OF TWO CASES OF GENERAL PARALYSIS WITH FOCAL SYMPTOMS.

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Case I. A well-educated business man, 50 years old. One brother is an epileptic. No other heredity is known. No serious previous diseases. He had, however, a fall from the rigging of a boat three years before the onset of the present illness. He was unconscious for a short time, but had no other known after-effects. A history of syphilis could not be obtained, but the only informant is the sister, and the patient has been away from home a good deal.

The history of the patient's illness could be gathered almost only from letters which he wrote to his sister. The following are extracts which give not only a fair idea of the development of the disease, but also of the difficulty which, even early in the disease, the patient experienced in expressing himself in writing.

February 15th, 1901.—". . . I go for my health (sc. to Cal.) and go as because I have to at once. My head has troubled me g continuous way until I could not continue my business. . . . I can't write much buct I can't."

He consequently went to California on February 18th, and there is said to have been somewhat irritable, but nothing else is known about him.

On March 31st, 1901, he writes that this is the first letter he has been able to write since he went to California. He gives

PAGE NOT AVAILABLE

stricken " with an attack of meningeal apoplexy and was for three days paralysed almost completely, including limbs, sphincters, sight, and throat." Two weeks later he writes: "The patient is able to walk, eats solid food, has almost complete control of sphincters, but *talks and sees imperfectly.* He can make his wants known; has been violent at times, but is now very pliable and easy to care for." (A history from the sanatorium could not be obtained, though asked for.)

Early in February 1902 he was brought to Boston, and arrived on the 6th. He was taken care of in an hotel, and, according to the information obtained, his state did not differ from that seen under observation.

He was admitted to M'Lean Hospital on February 9th, 1902. At the hospital, where he remained until his death on March 4th, the patient showed on the whole a stable condition until three days before he died. His general behaviour may first be sketched. This will be followed by a description of his aphasia and his disorder of sight, and the result of the general physical examinations.

The patient lay on his bed or sat in the chair, a good deal of the time apparently dozing, or sometimes he walked aimlessly about the ward, frequently bumping into objects. When anyone entered his room he jumped up quickly, and when the physicians entered, usually shook their hands vigorously, and at once began to talk rapidly and voluminously, but his talk was often meaningless. He had a small vocabulary, and used wrong words, yet short sentences were at times quite clear and correct. He had great difficulty in understanding what was said to him. (All these traits will be brought out more clearly later.) His mood presented decided changes. He often beamed with smiles, and generally gave the impression of decided euphoria. At times he laughed immoderately without appreciable cause. Again he got irritable during the visits, or more often, especially with the nurses, was easily fussed up, and once tried to strike a patient who took something from him at the table. Often he would not let the nurses take his temperature, once refused to undress at night, nevertheless he quite frequently appeared grateful and appreciative of what was done for him. As a rule he was tidy, but once defecated in his room.

He usually found his room in the ward, but again wandered into other patients' rooms. He was often unable to find the water-closet. When his people came he often recognised them. But again gave no clear evidence of knowing them.

On two occasions he seems to have had hallucinations; both times he sat looking into the register, and said something about people "down there" who were frightening him.

At the examinations he was, as above stated, often euphoric, again irritable, seemed to tire very easily, and on account of these features, together with his evident difficulty in understanding spoken language, his co-operation in tests was often very poor. But a very striking feature was his frequent evidence of insight. When he could not understand or could not find his words, his desperate "Oh dear," or statements like "I know but up here (pointing to head) I can't say anything," or his pathetic appeal once, "Oh, if I only could get them out of here! Won't you help me?" showed a certain appreciation of his defect, and argued strongly against any deeper degree of dementia. Sometimes he cried at these times, when he spoke of his condition; on the other hand, when, after desperate attempts to get a certain expression, he finally succeeded, he was childishly pleased. His *speech showed no articulatory disorder.*

The following extracts from the notes will illustrate his condition more in detail:—

Spoken commands.—

(Show your tongue.) Complies.

(Raise your right arm.) After puzzled thinking raises the left.

(Raise your left arm.) Puts up right.

(Sit back in your chair.) Straightens up.

(Lean back in your chair.) "What? What it's?" Then looks about puzzled, evidently thinking, finally leans back, saying, "You mean this?"

(Straighten your leg.) "Leg? What? That? (pointing to leg.) You mean straighten it?" Then rubs head vigorously. "This is it, worse, worse"; he continues to rub his head, finally says, smiling broadly, "Now I feel good."

In his daily life in the ward, according to the nurses, he sometimes understood and did as told, while at other times he seemed not to understand. On one occasion we told him re-

peatedly about an accident which befell some friends of his, but he was unable to understand.

Questions.—

(What is your name?) Pointing to head, "Here, can't you feel? Can't you find? Feel? There, that's what it is, see?"

(What is your name?) "What is my name now?" He could not be made to give it.

(How old are you?) "What say? Please? Call? What do you mean? What say? What call it? Say what?"

(How old are you?) "Co? No. I can't tell you." Then, irritably, "You don't tell."

(What is your age?) "What kind of work? I can't tell, she pulls me here something."

(Who is President of the U.S.?) "What's I am? Where I believe I am"; irritably: "I don't know anything about it. No, sir, I can't tell her anything about it. No, sir, can't tell. Something of that kind, see? We are good, you know. We have got the world, you see"; and, rubbing his head vigorously, "Cut that right off, pull it off, you know. There it is, see? See? There, it's a good question all the way. See? See? That's all the way, you see; that's all I can tell you, you see." (laughing immoderately). Then, putting his hand to his head again, "There is one thing that don't let me carry here. I want something front here in a piece, put here on me." Pointing to the physician: "Say, this man, this man has got it, it something there that is."

At another interview he was asked his name. He said, "Oh, well, sir, I have been there. F. something." Pulls a notebook from his pocket and turns the leaves, and says correctly, "Well, it's Frank, that is funny, if I should speak to you—Frank, F. I think it is Frank E. (correct). Well, I have been torn all to pieces (hand to head). But here now I am all mixed over again, I have been and had to had."

(Where are you?) "I do not know anything at all. I don't know about these things, but if you let me know I will try. Oh, I want to go and rest, and then I'll tell you" (indicating the bed. closing his eyes, and turning head sideways).

Naming of Objects.—

(Hat.) "Now, I want something like that." Takes the hat and handles it. "That tells you more than anything else."

(Handkerchief.) Looks at it intently. "What mean? Why, it's all right. It's something that. Let me go now, see?" He leans back, closes his eyes, saying rather despairingly, "I can't tell you anything now."

(Watch.) Points to it. "That? Carren a gold." (What is it?) He points to the face of the watch, and says, "6—10, about call 10" (it was 12). (What is it?) "What's this? Is leaf, leach."

(Cigar.) "What is—oh, that—that's a———." Puts out his tongue and wets his lips, then in an irritated manner, "Oh," and eagerly, "Hold it up a minute. I think that's called go—go—here this way———."

(Keys.) "I don't know a thing. No, I can't call it. No, now I have got something here. I don't know what it is. He is a good man" (not pointing to anyone).

(Ten cent piece.) "Coin, see? Of some kind. We don't carry that, don't we? That's another kind (fumbling it). We have one of our own." Then turns to the physician, "I'll tell you everything I can think, but sometimes I can't think. You see I can't reason." Tapping his head: "Where is something? If I had something I could see in here."

According to the nurses he was most of the time unable to say what he wanted, but sometimes indicated his wish by signs. Thus he one day drew his finger around his neck and wrists when he was dressing, indicating that he wanted collar and cuffs, and was pleased when they were brought. On another occasion he pointed to his stomach and his mouth, and drew a square; the nurse brought him a lunch consisting of milk and crackers, and the patient appeared very pleased, saying, "That's it."

Usually he cannot be made to *repeat words*, but talks in his usual manner, but once repeated "waiting." For "alliance" said "lions," adding: "That's funny I can't tell you so now. I can't get any of them, I can't peek."

He could not be made to *write* even his name; nor could he, as a rule, be made to copy anything, but would point to the different letters, miscalling them. When at one time he had, after much difficulty, read his name, and was told to copy it, he did copy the first two letters as if drawing them, and added the others in writing correctly, though awkwardly, and then appended

an "m." He noticed it was wrong and seemed worried about it, but could not improve it.

He was totally unable to *read* even individual letters (large print). Instead of reading he pointed to the individual letters, invariably miscalling them, and was unable to recognise them when they were directly shown and even named, and any *written commands* were treated in the same way and never complied with. Only once did he read correctly, namely when an envelope was shown him and he read "Doctor." His name in large print was given to him at another interview. He pointed to it and said, "L—e—this would be—l—e—e—what's that for? To please you or to do anything for you—l—e—let's see—e—l—I mean so as to make it look good. L—e—o—you see that don't look well—to make it something—to give it some knowledge," etc. Finally he gave his name (which has L and e in it), but did not recognise it, repeated it several times, varied it, looked discouraged, but finally with marked pleasure said, "That's my name. I'm a L . . . that's it."

In regard to his ability to *know the use of objects and to execute acts*, it should be mentioned that he handled spoon and fork correctly, attended to his own toilet and put on his clothes in a correct manner.

Of the physical examination the following may be mentioned: The eyes did not move equally, but the patient co-operated too poorly to examine them well. However, when he looked towards the left, the right eye did not move as far to the left as did the left. On other examinations it was noticed that when the right eye looked directly at the physician the left was turned outward. (The sister stated that the patient had been cross-eyed for ten years.) The pupils were normal in size, equal and even in outline. The right pupil did not react to light, while the left reacted normally. When the patient looked about, the width of the pupil changed. In tests for accommodation he did not sufficiently co-operate. The face was equally innervated. The tongue was protruded straight. There was no tremor of tongue, facial muscles, or extended fingers. There was no unsteadiness in walking. Both grips were equal, though not strong (co-operation?). The patellar reflexes were prompt on both sides, but the right distinctly more so than the left. On the right side there was also a more active tendo Achillis reflex and a few

rebounds of an ankle clonus. The plantar reflexes, however, were equal, and consisted in a downward motion of the toes and pulling up of legs. Sensation, so far as rough examination with pin pricks went, without good co-operation, seemed not interfered with.

Vision.—That there was a defect of vision has been stated in the history. It will be remembered that one of his physicians wrote that, after the apoplectiform attack of January 17th, the patient had a disorder of sight. It was also noted above that the patient often bumped into objects, yet I could never assure myself that this tendency was greater on one side than on the other. It was difficult to determine the nature of this disorder, owing to the poor co-operation on the part of the patient. Sometimes he did not react to stimuli coming from either side until they reached the centre of the field of vision. This was the case when objects were brought from the lateral side of the field of vision, while the physician stood behind the patient, so that there was nothing but the object itself to attract his attention. But when the physician sat in front of the patient and with gestures made him understand that he wished him to grasp an object brought into his field of vision from the outside as soon as he saw it, it was found that he would do so on the right side only when the object traversed the median line, whereas on the left he repeatedly grasped it at an angle of from 50 to 55 degrees. There seemed, therefore, to exist, even on the left side, a certain defect, inasmuch as the patient's attention could not be attracted by objects in the extreme lateral portion of his field of vision, while on the right side this evidently amounted to a half-sided defect. It was, unfortunately, impossible to get more accurate data.

On March 2nd he was found leaning on his bureau; he seemed to have difficulty in moving, and when the nurse assisted him to bed he is said to have appeared frightened. Soon his right arm and hand began to twitch, later also his right leg and the right side of his face. When seen by a physician the twitching had almost subsided, but the right hand, which lay on the chest, appeared tremulous, and was at times jerked towards the mid-line and upward. He could lift the right arm, but not grip the physician's hand. The right leg was rather flaccid, but not totally paralysed. He remained conscious, and spoke, but his

speech was even more confused than it had been. The twitching continued for twenty hours with varying intensity, and although in the night, at times, both sides were implicated, it was still much more evident on the right side; the face, however, was but little affected. At the end of this time, 10.30 A.M., on March 3rd, he became unconscious and had a general convulsion. He was found lying on his back with his head and eyes turned to the left, the latter also somewhat turned upward. Both sides were flaccid. Patellar reflexes equal and active. The two sides of the face equal. He was breathing stertorously and reacted in no way. While the physician was still present a second convulsion came on; it began with flexion and inward rotation of the right foot, followed by a slight raising of the right leg; then the same motions began in the left leg. Soon the quadriceps began to contract violently on both sides, while at the same time both arms were flexed, the right more than the left, the hands became flexed at the wrists. There was a tetanic spasm seen in the affected groups of muscles, with slight shocks, which soon became more pronounced, and amounted to about 132 a minute (not all synchronous with the pulse). The right side of the face twitched. Finally the left side of the face and the left arm became more affected; the latter was held in the air, the forearm and hand flexed somewhat, and showing frequent quick contractions; these were also seen in the left pectoralis.

At 11.5 he had a third convulsion, and from then on they recurred at frequent intervals, but varied in intensity. They were then distinctly more marked on the left side. This continued until 5.45 A.M. on the 4th, when he became comatose, the twitching stopped, he breathed stertorously, his face was deeply cyanosed, the eyes turned upward, purple blotches were seen all over the body. The temperature had reached 102·8°, pulse 130, respirations 52. He died at 2.46 P.M. on March 4th.

At the *autopsy* it was found that the left hemisphere was smaller than the right. The pia was thickened over the entire anterior portion of the brain; the thickening showing no special difference on the two sides, however. After hardening in formalin the left hemisphere appeared smaller, especially in the lateral diameter; whereas in the longitudinal and vertical diameters the difference was much slighter. On the convexity the convolutions of the left side, except those over the posterior part of the hemi-

sphere, were smaller than on the right side. The difference was extremely marked in the case of the first temporal convolution. This convolution appeared unusually narrow on the left side. The difference in weight of the two hemispheres amounted to 120 grammes in favour of the right; the respective weights being 710 and 590 grammes. On longitudinal sections the lateral ventricles presented no notable difference, but the left thalamus appeared somewhat smaller than the right. The two cerebellar hemispheres were the same in appearance and weight. The base of the brain and spinal cord presented nothing of note.

The following regions were examined, chiefly with Nissl's stain and by means of Benda's neuroglia method: both first frontal convolutions, both paracentral lobules, both first temporal convolutions, and pieces from the region of the fissura calcarina on either side. Later some large horizontal sections were made and three levels of the spinal cord examined.

Microscopical Examination.

The left first frontal convolution and the left paracentral lobule present the typical lesions of general paralysis in a pronounced degree. With the low power the whole cortex appears markedly narrower than that of the corresponding pieces from the right side, the cells are closer together, the general architecture is considerably disturbed. The blood-vessels are prominent, appear increased in number, and present small-cell infiltrations around them.

With the high power it is seen that among the cells in the adventitial sheaths there are numerous typical plasma cells.

Rod cells, such as Nissl and Alzheimer have described, are found in the tissue in considerable numbers. The glia shows marked changes, many nuclei are rather large, and well-defined cell bodies are often seen.

The nerve cells show various degrees and forms of alteration.

The changes in the corresponding regions of the right side are much less marked, though by no means absent. The process in the right first temporal convolution is of about the same intensity as that of the first frontal convolution and the paracentral lobule of that side, whereas, in the region of the right calcarine fissure, the changes are very slight indeed. Here the disorder of the architecture is almost absent, but the glia shows

some changes, and some plasma cells are found in the vessel sheaths.

We find a very different picture in the left first temporal convolution, and in the region of the left fissura calcarina.

The left first temporal convolution.—Even to the naked eye the cortex looks very narrow, and the tissue cuts less well than in a normal cortex. With the low power it is at once apparent that the architecture of the cortex is grossly disordered. Many nerve cells have disappeared, while there are present large, pale cells in considerable numbers. The blood-vessels are less conspicuous than in the less-affected portions of the brain, and the perivascular infiltrations slighter. With the high power the glia shows marked changes. The pale cells above mentioned have large, greyish-blue cell bodies of a rather compact appearance. The outline is either uneven, showing the beginning of more or less numerous processes, or the outline fades imperceptibly into the surrounding tissue. These cells either contain a large, pale nucleus, or a small, irregular one. The nuclei are very apt to be eccentric. In the most affected areas the vessels, as has already been stated, are not prominent. The cell infiltration with round cells and plasma cells is much less marked than in the less affected regions. Moreover, the vessels seem fewer in number, and rod cells much less frequently met with than in the portions of the cortex where the process was apparently more acute. The nerve cells show very marked alterations. The sections stained with Benda's neuroglia method were not as successful as is desirable, since the neuroglia fibres did not stain as well as they should. Nevertheless these sections are instructive, since the cell bodies of the neuroglia cells are stained very well, so that with the low power a very good idea may be obtained, not only of the marked disorder of the general architecture of the cortex, but of the large number of these cells (Fig. 1). Fig. 2 does not give an adequate impression of the cells as seen with the oil-immersion lens, since an attempt was made to get the best general picture on the photograph, rather than to focus upon a special cell. In the sections many of the cells appear very large, presenting a homogeneous cell body, and often quite broad processes. In many places the cells present, at the periphery of the cell body or of the processes, rather coarse, deeply-stained fibres. In other cells the entire process is deeply

stained, and it is impossible to clearly demarcate the fibres. Finally, there are cells and processes which present no differentiated fibres at all. Free fibres are also seen in the tissue. These cells are present throughout the cortex, and are also quite numerous in the white matter.

The changes just described are those in the most affected areas. In other sections they are less pronounced, and even in the same section the intensity of the alteration varies in different parts.

Fissura calcarina.—The picture in this region does not differ from that just described, but here the changes appear to be more patchy. Fig. 3 shows that there is a more normal strip going horizontally through the middle of the cortex, whereas, in the upper and lower layers, the changes do not differ in intensity and character from those described in the first temporal convolution. Fig. 4 shows the large glia cells as seen with the high power. On the whole there are places where the entire cortex is almost as much affected as in the most implicated parts of the first temporal convolution, but the intensity seems to vary rather more, and the marked destruction seems less widespread.

Besides the small sections, I have at my disposal some large horizontal sections through both halves of the brain, which supplement well the findings in the former. These sections take in the tip of the first temporal convolution and the second temporal convolution, and the fissura calcarina. In these large sections the difference between the right and the left side is quite apparent, even to the naked eye, inasmuch as the entire left side is considerably smaller than the right side. The cortex, too, especially that of the first and second temporal convolutions, is much thinner on the left than on the right side.

On microscopical examination of the left side it is seen that the second temporal and, especially, the first temporal convolution are more affected than any other convolution at the convexity of the brain. The first temporal presents a marked increase in glia in places, whereas the rest of the cortex is chiefly conspicuous by the marked perivascular infiltrations which are present everywhere. Quite striking are the changes in the fissura calcarina so far as it is struck by the section. Even with the low power large cells are seen, which, with the high power,

can easily be identified as large glia cells such as we described in the picture obtained from the small section. This condition is seen especially in the first and second layer of the cortex, but dips down in a varying degree in different parts. The vessels are not prominent and the perivascular infiltrations slight.

On the right side the changes are more marked in the entire frontal lobe than in the temporal region and the parts behind this, so far as the convolutions of the convexity are concerned. The changes seem to be of moderate intensity as judged from the perivascular infiltration. At the tip of the frontal lobe there is a considerable area over which there is seen, especially in the deeper layers of the cortex, a marked growth of large glia cells, so that here an old process was evidently located, which, it is fair to assume, occurred at the same time as the other more pronounced alterations, namely, at the time of apoplectiform attacks. The finding in this region shows how difficult it is to judge of the distribution of the process from the small sections only. This applies equally when we come to consider the right calcarine fissure, which was free from any change in the small sections. In the large sections we here find a condition similar to, though less intense than, that on the left side, namely, a marked overgrowth of glia in the shape of large cells, seen more especially in the first and second layer, but also dipping down deeper in some places. The blood-vessels in this region, except those of the pia, are not conspicuous, and in this way this region also resembles that of the opposite side, and the other areas in which the process had been more destructive. The optic radiations are apparently intact. The external geniculate bodies are unfortunately not in the plane of the sections.

An examination of sections from the cervical, thoracic, and lumbar cord revealed a bilateral degeneration of the pyramidal tracts of moderate degree and of the same intensity on the two sides. This degeneration was not present in the thoracic and cervical region.

Summary.

We have, therefore, a case of general paralysis which commenced somewhat over a year before its termination, probably with a mild apoplectiform attack ("I was stupid and could not use my tongue"). This was followed by an aphasic

disorder which seems to have gradually improved. There were severe headaches. There seems to have been no decided evidence of a general mental involvement. A year after the onset the patient had a marked apoplectiform attack which left him in a condition as follows:—Probably moderate dementia, certainly euphoria and irritability, limited vocabulary, paraphasia, loquaciousness, great difficulty in understanding spoken language, great difficulty in reading and writing. One pupil inactive to light; reflexes increased on the right. Right-sided hemianopsia and difficulty of vision on the left side.

At the autopsy the left hemisphere was smaller than the right (difference, 120 grammes). Changes in the cortex throughout, more marked in the left hemisphere. Profounder changes denoting a less active process in the left first temporal convolution and region of the left fissura calcarina. Some, though less extensive, changes of the same character in the right calcarine fissure and at the tip of the right frontal convolution. Bilateral pyramidal degeneration in lumbar cord.

The interpretation of this case seems to be this:—Early in the course there took place a fairly intense process in the left temporal lobe. This was later followed by another process of a more destructive character, again localised in the first temporal convolution, but also in both calcarine fissures, especially that of the left side. Such processes were first described by Lissauer, and later by Alzheimer. As is usually the case, these more intense processes gave rise to apoplectiform attacks with subsequent focal symptoms. The more generalised process in which the inflammatory changes were more in the foreground was also more marked on the left side throughout.

The sensory aphasia finds its explanation in the changes situated in the first temporal convolution. The visual disorder is especially interesting. Though it was bilateral, the patient was not blind, but often bumped into objects on either side, and it was found by tests that on the whole right side his attention could not be attracted by movements of the hands or by objects; whereas on the left side this was difficult and could only be done to a limited degree, so far as extent and intensity were concerned, namely, only when the stimulus was expected and obtrusive, and even then not farther out of the direct vision than to about 50 or 55 degrees of the temporal side of the field. It

is fair to assume that this disorder of sight is due to the changes which we could demonstrate in both calcarine fissures.

CASE II. A man 37 years old. So far as the family history is concerned we find a marked psychopathic heredity. Since early youth abnormal traits were noticed in him: he was untruthful, boastful, and when he grew up became very dissipated. In 1888 he had syphilis.

In 1899 the patient began to lose weight, complained of fatigue, and at times of dizziness. At the beginning of 1900 his normal tendency to bragging became a little more marked. In May 1900 he had a series of convulsions lasting for five hours. These left no sequels and he was not markedly worse in any way. Three months later (August 1900) he had another series of convulsions, after which one leg is said to have been paralysed, the other weak. (The informants could not state which side was the one more affected.) He also was unable to speak and write. This passed over in a short time. He was sent to a sanatorium, where he is said to have had Argyll-Robertson pupils, a slight paralytic speech defect, grandiose ideas, and marked incoherence. In January, when again at home, he had convulsions and was unconscious for four days. This left him with weakness in the right arm and hand, so that he could not hold objects well. Subsequent to this he had occasional attacks of twitching in the right arm and hand, and on several occasions these became general. These attacks were not associated with loss of consciousness. This condition of paresis of the right arm with attacks of twitching in it persisted for four months. His speech at the time was thick, but he was evidently not aphasic. His mental condition was one of euphoria and irritability, but it is claimed that he often spoke intelligently and was not markedly weak mentally. Then, suddenly, three weeks before admission (middle of May 1901), he became pale and dull, and it was found that he was paralysed in the right arm and that he had lost his speech. He became "semi-conscious." Two days later the right leg became paralysed. He remained speechless for a week. After that he was able to utter some monosyllables, while the paralysis of his leg improved, but not that of his arm. Ten days after the onset of these symptoms he had twelve convulsions. During three days he was unconscious. The convul-

sions were chiefly localised on the right side. His speech was again totally lost. The weakness of the right arm and, to a lesser extent, that of the leg persisted. He is said to have been very irritable and at times euphoric.

The patient was at the hospital about three months. During this time his general mental condition as well as his aphasic disorder changed very little. For the sake of simplicity we may therefore summarise this and then speak of the other features, more especially of the condition of the right side, more in detail and in chronological order. The patient often seemed dull and his attention was attracted with difficulty, and he frequently seemed not to understand what was said to him, though at other times he executed some commands, but usually co-operated very poorly. At times he would put out his tongue when told, though usually he did not; once after he had correctly complied with this request and was then repeatedly told to take hold of a pencil, he finally again protruded the tongue. Again, he could a few times be made to attempt the naming of objects; often it seemed impossible to make him understand what was wanted. In sensory tests and tests for hemianopsia there was very little co-operation.

He was unable to speak, and, as a rule, only produced senseless syllables like "pur pew" when speaking spontaneously, though we were told that a few times, when his people were with him, and when he was evidently under some emotion, he spoke better, and on one such occasion said to his brother, as he was leaving, quite clearly, "I want you to stay," again, "Good-bye, Willie," or once, "Mother"; but these examples were quite isolated, and in the presence of the physician nothing like it was ever obtained from him. The nurse reported that on one occasion he sang the doxology so that the melody could be caught, but that the words were merely the usual senseless syllables. Of the results obtained on the few occasions when it was possible to make him attempt to name objects, the following samples are typical. When told to name a watch he said clearly, "watch"; immediately afterwards for handkerchief, "hatched—che—che—pil—pil—watch—watch." For dollar bill, "watch—watch—you say watch—and some zitch—witch." On another occasion he said for watch, "wash—bosh—pur"; for dollar bill, "atchee—pur—pur";

for keys, "hatchee—pur—pur—la." Once he was able to give his first name, and when several ages were given he picked out the correct one. At other times he did not react to all such tests. He usually could not be made to write, not even with the left hand, but put the pencil down, when it was put in his hand, without making an attempt, or did not even take the pencil. But on one occasion, early in August, he wrote, of his own accord, his name correctly with his left hand, but seemed unable to write the r of Jr. A number of times an attempt was made to make him read. At times he did not react at all, at other times he produced the same senseless syllables as in speaking, though once pronounced a few words, as "and," "the," "don't," "do," correctly.

As to his general mental condition, it may be stated that he had to be cared for a great deal, not only on account of the paresis of his right arm (to be presently described), but chiefly on account of his mental condition. He frequently was untidy, though he also often went to the chair when told to. He sometimes fed himself with the left hand, again had to be fed. At times he had a tendency to chew insufficiently, so that his food had to be cut up into small pieces lest he choke. Frequently he was decidedly euphoric, would laugh and grin a great deal, make frequent attempts to speak, at times punch the physician playfully; but even on these apparently brighter days his co-operation was but little better, whether the commands were given in gestures or spoken language. Again he was very irritable, would even throw his dishes on the floor, or he tossed restlessly about the bed, getting angry when anything was done for him. Sometimes he cried.

There was at no time any evidence of hemianopsia; when he walked about he never bumped against objects. Tests were quite unsatisfactory, owing to lack of co-operation.

Besides his aphasia, the most striking condition was that of his right side, especially his arm. For the first ten days the arm was used but little, and when he could be made to touch the physician's hand, he would raise his arm and, with a swaying and wavering motion, bring it towards the hand of the examiner, miss it, try again, miss it again, until he finally reached it. He could not be made to grip the hand. These ataxic motions were present whenever he was seen in this

period. The arm or hand presented no atrophy at this time or later. He was able to walk well, and seemed not at all one-sided. Knee-jerks, tendo Achillis, and plantar reflexes were normal, and there was no difference on the two sides. The cremasterics were sluggish. Four days later, the reflexes were a little more active, but still equal. The face showed no asymmetry at first, but five days after admission the right side showed distinct, though slight, paresis. The tongue was protruded straight, and showed no tremor. The pupils did not react to light; that there was normal accommodation could be seen by watching his eyes as he looked about (no co-operation). The eyes seemed freely movable, and there was no nystagmus. Sensory examination was quite unreliable; he paid no attention to pin pricks anywhere, except when pricked on the face; but there was no difference on the two sides.

On June 17th the nurse reported that while walking about the patient suddenly appeared pale, his eyes became staring, and his right arm began to twitch, but he did not lose consciousness. When seen a few minutes later he was rather duller than usual; the right forearm was in constant athetoid motion. The arm became pronated and supinated, while the fingers were flexed and extended, these motions being quite slow. They continued for ten days almost uninterruptedly; he could move his arm voluntarily, and it showed the same ataxia as before. The reflexes were obtained with less readiness. The facial paresis was not altered, the tongue was protruded straight.

On June 27th he had two short attacks, with rapid twitching of the whole right side. After that the former rotatory movements ceased and the arm appeared more useless, the right leg dragged a little, the face was the same as before.

On June 29th he became very dull, and showed some twitchings of the right arm and facial muscles of the right side, but not of the leg; he was found gripping the sheet tightly with the left hand, and groped aimlessly about when this was pulled away. The right arm did not fall quite limp when raised; it showed a slight resistance when passive motions were made, while the left resisted strongly. Both legs showed the same slight resistance. The knee-jerks were rather less active; the left rather more marked than the right.

From that time on the arm improved, but the condition varied; he used it at times more, again less freely; sometimes the grip was quite strong, again the same ataxic motions were seen as before. The right leg was now not different from the left. The facial paresis was unaltered, but the tongue now also deviated somewhat to the right. On a sensory examination made on July 24th, on which day the co-operation was a little better, the reaction to pin pricks was quite prompt, and distinctly better on the right than on the left side.

On July 31st he had three convulsions of short duration: in the first the whole body was implicated; in the two others the twitching was confined to the right arm and right side of face, while the eyes turned to the right. He had lost consciousness. After he regained consciousness the twitchings of the right arm and leg continued for some time; later, only the thigh twitched, while he appeared a little duller than usual. This was interrupted by a short attack in which his whole body stiffened, and on August 4th he was almost stuporous for a day. The arm was again more useless, and fell limp when raised; the leg was less limp, the facial palsy more marked than it had been. He got brighter on August 9th. The hand was now held with fingers flexed, and it was somewhat difficult to overcome this flexor spasm. This is not mentioned in later notes. It was then that for the first time the knee-jerks were more marked on the right side, but both were exaggerated. The plantar reflex was not obtained on the right side, but was normal on the left. The paresis of the right side did not improve after this. The patellar reflex remained greater on the right side; the plantar reflex on the right side was uncertain, though prompt on the left. There were occasional twitchings of the arm; little voluntary motion. On August 24th he became unconscious; there were marked convulsive motions on the right side which soon became general, but these lasted only a short time. A few hours later, Cheyne-Stokes breathing appeared, he did not regain consciousness and had no further convulsions; his respirations rose to 40, temperature not above 100°, pulse 140, and there were signs of consolidation at the base of the right lung. He died on August 26th.

At the autopsy the brain was asymmetrical, the left hemisphere being smaller than the right, especially in its lateral

diameter. The pia was thickened over the entire anterior portion of the brain. The tissue of the left Rolandic region seemed softer than that of the corresponding portion of the right side, and decortication was more marked. No attempt was made to strip the pia over the entire brain.

After formalin hardening, the left hemisphere appeared smaller than the right, chiefly in its lateral, to a lesser extent in its vertical, and to a still lesser extent in its longitudinal diameter; the shortening of the latter appearing only at the posterior portion of the hemisphere (difference 1 cm.). The right hemisphere weighed 647 grammes, the left 535, a difference of 112 grammes. On section, it was found that the left ventricle was somewhat dilated and extended further back than the right.

The right cerebellar hemisphere looked somewhat smaller.

The left internal carotid artery was smaller than the right.

From the following regions pieces were taken, and examined chiefly by means of Nissl's stain and Benda's neuroglia method: the left and right first frontal convolution, left and right paracentral lobule, left and right anterior central convolution, left and right first temporal convolution, left and right angular gyrus, left and right calcarine fissure, and left third frontal convolution. Furthermore, sections from the medulla and from three levels of the spinal cord were examined by means of Weigert-Pal, and adjoining pieces of the cord and a piece from the pons were treated with Marchi's method.

On microscopic examination of the sections from the cortex, the most conspicuous feature is, that in certain regions, namely, the left paracentral, the left anterior central, the left first temporal, and the left angular gyrus, a band of very large glia cells is seen in the fifth layer and the transition zone from the fifth layer to the white matter. This band is wide enough to cover $\frac{1}{2}$ to $\frac{3}{4}$ of the field when looked at with Ocul. 2, Object. 3, Leitz. The glia cells in this region are of the same character and the appearance of the tissue is the same as described and illustrated in Case I. The cells are again best seen in Benda preparations, in which the cell bodies are very prominent. The width of this band of glia overgrowth is about the same in the different regions mentioned; and in all of them, except in the paracentral lobule, the profound alterations are seen, in places, to extend further up into the cortex. In the fissura calcarina of the left

side a similar, but much smaller, band is seen. In all the other pieces, namely, all those taken from the right side as well as in the left first frontal and left third frontal convolution, there may be found fair-sized glia cells, but neither are they of the size, nor do they occur in such numbers as in the regions described. The difference is very striking with the low power. In Nissl sections these large glia cells are also quite prominent.

Apart from this glia overgrowth, we find almost everywhere in the cortex the typical alterations of general paralysis, viz. disorders in the architecture, perivascular infiltration with lymphoid cells and typical plasma cells, progressive and regressive changes in the neuroglia cells, new formation of blood-vessels, and the existence of more or less numerous rod cells. But the changes are not everywhere of the same intensity. In the first place, the perivascular infiltration, especially that about the larger vessels, is throughout more conspicuous in the sections taken from the right hemisphere than in those taken from the left, at least so far as the anterior central, paracentral, first temporal convolution and angular gyrus are concerned. The first frontal convolutions on the two sides present in this, as well as in every other way, the same picture. The calcarine fissure also takes a separate place, inasmuch as the right side is not affected at all, whereas the left side shows moderate changes. But while, as we have just stated, the perivascular infiltration is greater in many places of the right side, the other tissue changes are rather greater on the left side. Not only do we here find the band of glia overgrowth already mentioned, but even the upper layers of the cortex show a greater disorder of architecture, more marked changes in the glia, and, on the whole, a greater number of rod cells than on the right side; and the cortex is thinner on the left than on the right side.

It would appear from this, then, that while the frontal regions, so far as examined, are not different on the two sides, there is a marked difference behind the frontal regions. This shows itself, not only in the greater atrophy of the left hemisphere and the widening of the left ventricle, especially in its posterior part, and a shortening of the posterior part of the left hemisphere, but also, microscopically, in greater general tissue changes on the left side, and especially marked alterations in the fifth layer of the cortex and the transition zone to the white matter.

Considering the marked affection of the motor area, it is interesting to note the changes which are found in the brain stem and the spinal cord. In Weigert sections there is seen a moderate, though easily recognisable degeneration of the left pyramidal tract in the medulla oblongata (level of the olives). In the spinal cord it is impossible to discover any definite degeneration in the pyramidal tracts or any difference on the two sides. With Marchi, on the other hand, we find degeneration in both pyramids in a piece from the pons and in pieces from the cervical, thoracic, and lumbar regions of the spinal cord. In the pons this Marchi degeneration is very marked on the left, but slight on the right side. In the cord the pronounced Marchi degeneration can be traced to the left direct and to the right crossed pyramidal tract.

Summary.

We have, therefore, a case of general paralysis, who, after a rather indefinite beginning, had convulsions, followed by transient paralysis in one leg and aphasia. After that, Argyll-Robertson pupil, paralytic speech defect, euphoria, grandiose ideas, and incoherence are noted. In January 1901 he had convulsions again, followed by weakness, and, later, occasional attacks of twitchings in the right arm. In May 1901 there was a greater loss of power in the right arm, and again aphasia. The aphasia henceforth remained practically unchanged, whereas the condition of the right arm underwent various changes, and at times the right leg and face were affected. Under observation the right arm presented at first ataxia, then, after a slight apoplectiform attack, athetoid motions. The latter ceased after renewed attacks and gave place to twitching, while the arm became more helpless. With repeated attacks of the same kind the weakness increased, and an increase of reflexes developed on the right side. The aphasia was characterised by an almost total inability to speak and write, and, evidently, by a marked interference with the understanding of spoken language; but there was also a marked degree of dementia. At the autopsy there was found a marked general paralytic process, with a greater involvement of the left side, and, so far as the examination went, with especial involvement of the anterior central and first temporal convolutions and the angular gyrus, in which the

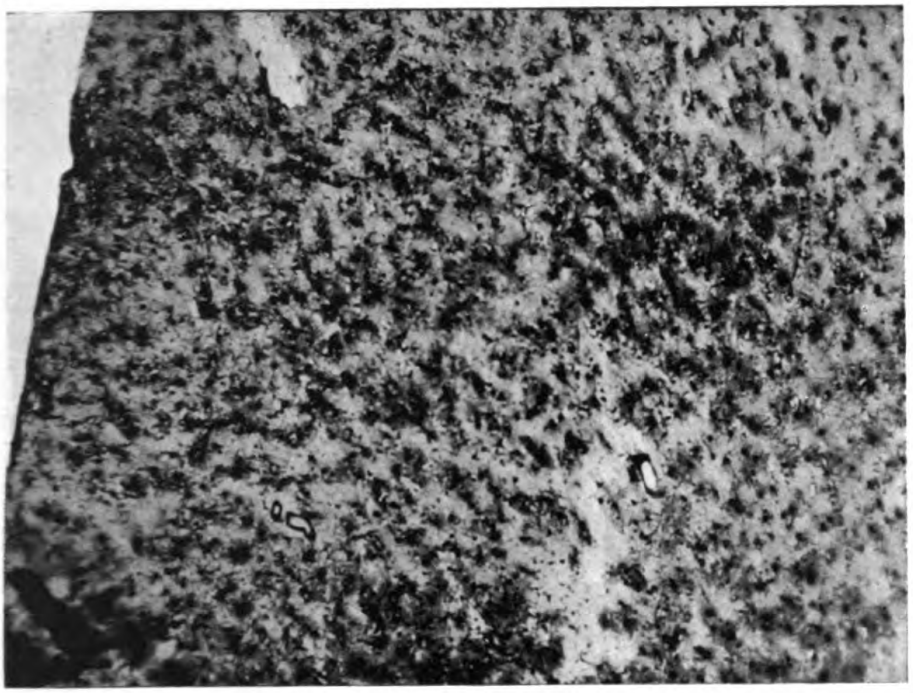

FIG. 1.

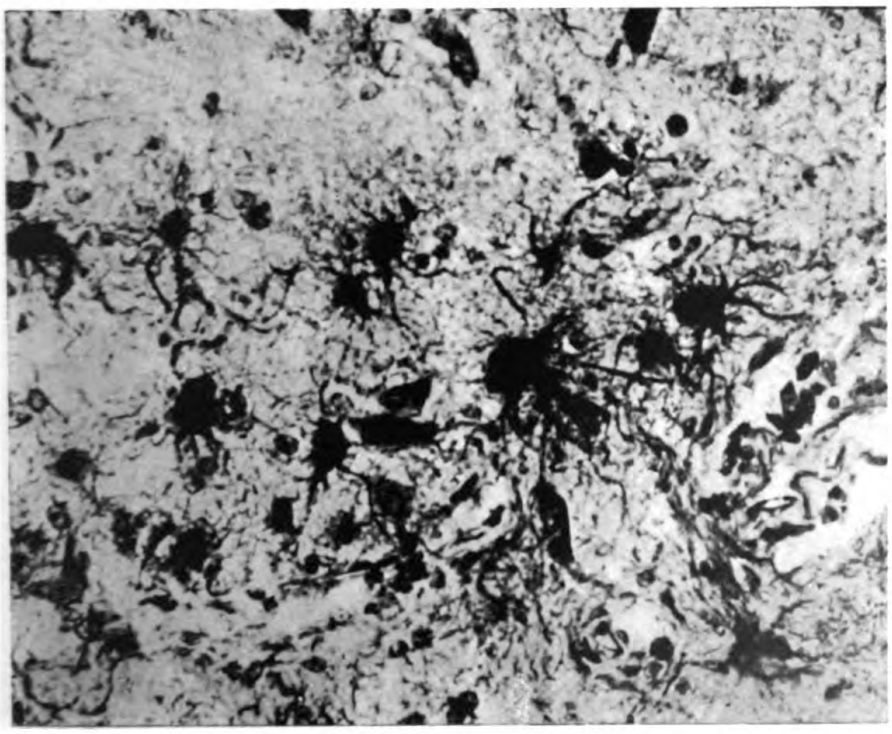

FIG. 2.

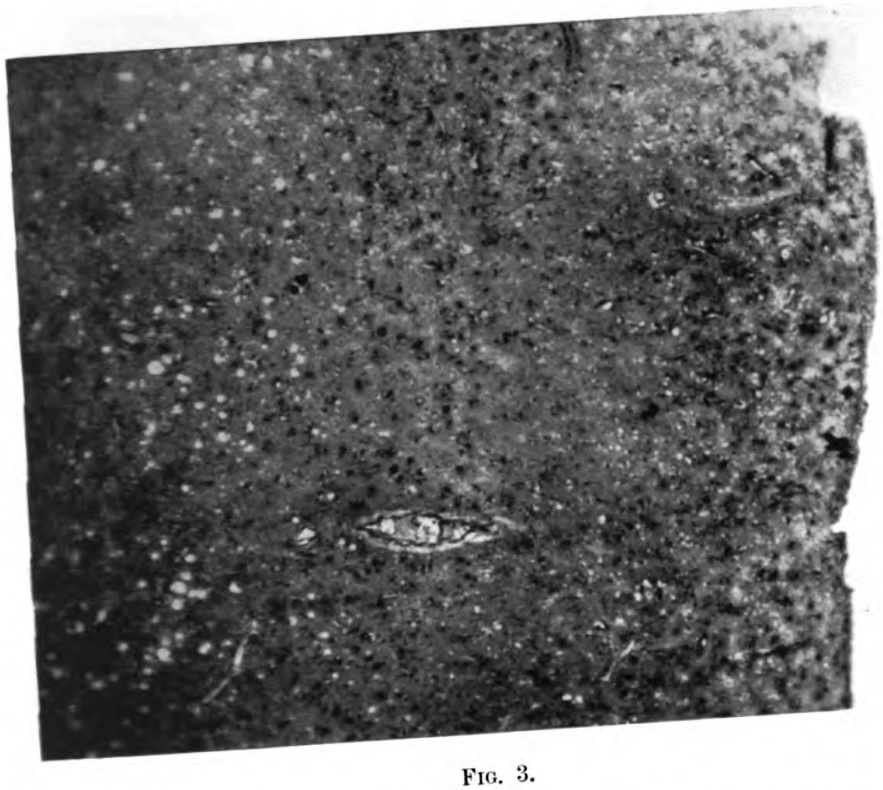

FIG. 3.

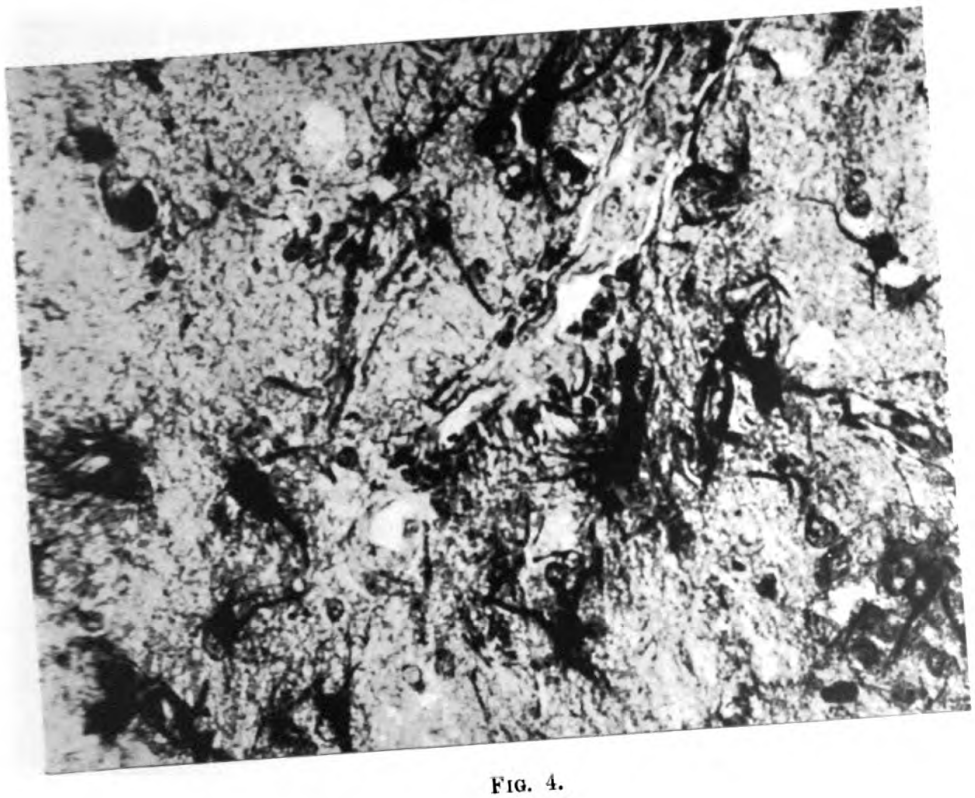

FIG. 4.

most marked feature was a broad band of glia overgrowth in the deeper regions of the cortex. There was also found a Marchi degeneration in both motor tracts, but much more intense in that coming from the left hemisphere. In this case the difference between the more affected and less affected areas is less clean cut than in Case I. The same may be said in regard to some of the clinical symptoms. The dementia was much more advanced, the aphasia more difficult to interpret. But it is interesting to note in this connection that the third frontal convolution was not among the more involved areas, but that the first temporal and the angular gyrus presented very intense changes. Of interest are the ataxia and athetoid movements and the attacks of twitching in the right arm, which symptoms were associated with and preceded the more marked loss of power.

Description of Plates.

Fig. 1.—Section from first left temporal convolution of Case I. Benda's neuroglia stain. Low power. Marked disorder of architecture. Disappearance of many nerve cells. Many large glia cells. Lack of prominence of vessels and of perivascular cell infiltration.

Fig. 2.—Section from first left temporal convolution of Case I. × 375. Benda's neuroglia stain. Showing detail of Fig. 1, especially the large glia cells.

Fig. 3.—Section from region of fissura calcarina in Case I. Low power. Benda's neuroglia stain. Showing similar condition as Fig. 1, but more patchy.

Fig. 4.—Section from left fissura calcarina of Case I. × 375. Benda's neuroglia stain. Showing detail of Fig. 3.

Abstracts

ANATOMY.

THE VALUE OF THE MYELOGENETIC FIELDS OF THE CEREBRAL CORTEX. (Der Wert der myelogenetischen Felder der Grosshirnrinde.) OSKAR VOGT, *Anatom. Anzeiger*, Bd. 29, 1906, p. 273.

(1)

THIS paper contains a vigorous assault on Flechsig's division of the cerebral cortex into forty or so different centres, according to the date of the myelinisation of their fibres. It is argued that Flechsig's method of subdivision is inferior to that obtained by the study of the arrangement of the myelinated fibres in the adult brain. The areas of the cortex which myelinate early are those which in the adult are characterised by the number and large calibre of their fibres. The comparison of Campbell's figures with the process of myelinisation in the developing brain shows that there is a complete parallelism between the richness of any region of the cortex in myelinated fibres and the date of the first appearance of myelinated fibres in that region. For instance, Campbell's areae precentralis et postcentralis, which are distinguished by their wealth in fibres, and especially by those of large calibre, correspond to the regions in which medullated fibres first appear. Similarly, a second focus of early myelinisation coincides with Campbell's visuo-sensory area. Although the results of the myelogenetic investigation of the cortex are in this connection significant, there is nothing to show that the anatomical division of the cortex founded upon it is applicable to the adult brain; on the other hand, the study of the arrangement of the myelinated fibres in the developed brain reveals all the peculiarities that each centre has acquired during the course of its development: it can, for example, distinguish between the structure of the precentral and the postcentral gyri, which was not possible from a study of the myelogenesis alone. Finally, as myelinisation starts in a certain number of foci, and spreads gradually from them, it is impossible to mark out such definite fields as Flechsig has attempted to do.

The question, however, arises: Are there anatomical and functional differences between the early and late myelinating areas, such as Flechsig assumes in stating that the former represent the sense, and the latter the association centres? There is no physiological proof of this, but Flechsig has urged two arguments in favour of it. Firstly, that the arrangement and character of the cells is different in the early and late myelinating areas, in that

there is a resemblance between the cell architecture of each of the sense centres and that of their corresponding receptive organs. But this is certainly not so. Secondly, that the later myelinating regions—Flechsig's association areas—do not possess any projection fibres. C. Vogt, however, has shown that portions of the projection systems remain non-myelinated as long as there are portions of the cortex unmyelinated, and that those portions of the projection systems correspond to those in which secondary degeneration is found after destruction of Flechsig's association centres in the adult brain. Therefore these too receive projection fibres. But Flechsig's attempt at the subdivision of the cortex by the myelogenetic method has not been valueless, even though his conclusions cannot be accepted; it has indicated that this organ contains both higher and lower centres, though these cannot be classified as projection and association areas as Flechsig has done.

On the other hand, there are strong indications that by a study of the different arrangementof the medullated fibres of the different parts of the cortex, areas can be mapped out that have different physiological functions. But the subdivision of the cortex is still more easily and more accurately possible by the study of the cyto-architecture of its gyri, as Campbell and Brodmann have shown.

GORDON HOLMES.

THE STRUCTURAL CENTRES OF THE CORTEX. (Ueber strukturelle Hirncentra mit besonderer Berücksichtigung der strukturellen Felder des Cortex Pallii.) (2) OSKAR VOGT, *Verhandl. der anatom. Gesellsch.*, 1906, p. 74.

FOR some years an attempt has been in progress in the Neurobiological Institute in Berlin to divide the cerebral cortex into structural centres, that is, into areas which can be distinguished by fibre systems, or by their specific cell arrangement. Such a division can be made on the basis of the character and arrangement of the cells, of the myelinated fibres, or of the non-medullated fibrils, and the results obtained by the application of these different methods must be correlated and brought into agreement. The study of the cyto-architecture has been found to be the most simple; by it areas of cortex are distinguished by the cell structure of their component layers, and by the arrangement of these layers. Vogt's aim in the present paper has been to determine, in the first place, the relation of these structural centres of the cortex to the areas which can be marked out as belonging to any function; and, secondly, to discover what relation structural centres bear to the sulci, which are our chief landmarks in anatomical localisation. His

data as to the structural centres are obtained from Campbell and Brodmann's publications, but for physiological localisation he has relied largely on the results of numerous experiments of his own on the localisation of the motor centres. It is interesting to see the marked similarity there is between the independently obtained conclusions of Brodmann and Campbell on histological localisation; many of the areas outlined by them seem to coincide accurately at least, in the higher apes, on which the greater part of Brodmann's work has been done. Further, Brodmann has succeeded, as Campbell had previously done, in following at least two of the centres—the precentral and visuo-sensory of Campbell—through a whole series of animals, from marsupials to man.

Two anatomical types have been chosen, the precentral and the postcentral, on which to study the relation of the functional to the structural centres. There is an abrupt change in the type of cortex in man, the anthropoid apes, and the lower monkeys, at or near the fissure of Rolando, and the evidence is accumulating that these different types have different physiological properties. (1) Anatomical evidence: It is generally accepted that the fibres of the pyramidal system arise only from the precentral gyrus, though Vogt has found very slight degeneration in these tracts after lesions of the postcentral area in the lower monkeys. Further, the only degenerated fibres which reach the thalamus after destruction of the precentral area enter the nucleus in which the tegmental radiation ends, while those which come from the postcentral area enter the nucleus of termination of the fillet. This fact also suggests that the precentral and postcentral areas have different functions. (2) Physiological evidence: Vogt, by using minimal currents in the orang, arrived at the same conclusion as Sherrington and Grünbaum, viz. that all the excitable motor areas lie in front of the central sulcus. In the lower apes, too (twenty-four animals), he found that all excitable foci lie at least 1 mm. in front of the fissure of Rolando. Further, the frontal limits of the excitable limb areas correspond accurately to the frontal limit of Brodmann's precentral type of cortex. The centres from which movements of the eyes and head, and the movements of mastication, can be obtained, correspond with further frontally situated structural centres of Brodmann. Again, in some of the lower apes the electrically excitable centres do not extend as far back as the fissure of Rolando, and in them the motor type of cortex has the same posterior limit. (3) Evidence obtained from destructive lesions: In the lower apes (11 animals), symptoms of palsy are obtained only after destruction of the precentral gyrus, and the movements which remain possible are well co-ordinated, while if the post-central is injured no palsy is observed, but there is considerable ataxia

of movement. Therefore, in the case of the motor cortex at least, the structural areas coincide with the physiological centres.

On examining one of the diagrams of Brodmann or of Campbell, it is at once apparent that many of the structural centres have no relation to the furrows, and that the margins of others are not bounded by the sulci. But Ziehen and Kükenthal have insisted on the constancy of the chief sulci, and maintain that the homology of these sulci is possible in all placental mammals. But it is evident that physiological centres do not occupy analogous relations to possibly homologous fissures, and it is highly improbable that the variability in the position of analogous cortical fields to the furrows is to be ascribed to a wandering of the fields. It seems more probable that it is the position of the furrows which is inconstant, and that analogous and homologous cortical areas can be regarded as identical. GORDON HOLMES.

THE FASCIA DENTATA AND ITS SUBCALLOSAL CONTINUATION. (Le Circonvolution godronnée et ses Prolongements sus-calleux.) (3) TROLARD, *Rev. Neurol.*, Oct. 30, 1906, p. 909.

ONLY macroscopical dissection was used in this investigation. The size and relations of the fascia dentata are first described; posteriorly it is continuous with the fasciola cinerea, which is composed of both grey and white matter. It is generally believed that the fasciola cinerea bends round the splenium, and forms the induseum griseum which covers the dorsal surface of the corpus callosum, as well as the striæ Lancisi and the tænia tecti. Trolard also admits that this may be so, but according to him its most important components are the small portions of grey and white matter which lie on the ventral surface of the limbic gyrus, and form what he calls the gyrus sublimbicus. This can be traced as far forwards as the genu of the corpus callosum; here the grey matter disappears and the white fibres enter the septum. These fibres have no connection with the cingulum.

GORDON HOLMES.

CEREBRAL TOPOGRAPHY AT THE SECTION TABLE. (4) C. B. FARRAR, *Am. Journ. of Insanity,* No. 1, 1906, p. 69.

THE cortex of different brains can be satisfactorily compared, either from the anatomical or physiological point of view, only by comparing approximately similar areas; and in routine pathological examination it is essential, not only that representative areas be chosen, but that in each case the same representative areas be selected. The author indicates, in diagrams attached to

his paper, five areas of cortex which may be suitably selected for such routine examination. These include portions of the central, frontal, temporal, calcarine, and hippocampal gyri, but each laboratory will probably prefer a method of routine examination of its own. GORDON HOLMES.

PHYSIOLOGY.

(5) **CHEMICAL STIMULATION OF THE MOTOR AREAS OF THE CEREBRAL HEMISPHERES.** S. S. MAXWELL, *Journ. of Biol. Chem.*, Vol. ii., 1906, No. 3, pp. 183-194.

FOLLOWING Loeb's rule that chemical stimulation can be best brought about through the action of those substances, whose anions precipitate calcium, the author employed chiefly solutions of citrates and oxalates in his experiments. The experiments were performed upon the motor areas of rabbits and cats.

If the substances were applied to the surface of the brain, no immediate response was obtained. Their effects were only noticeable after a latent period of many minutes (ten minutes or more).

If one or two drops of citrate and oxalate solutions, isosmotic with blood serum, were injected into the brain substance by means of a specially constructed hypodermic needle, either no response or an immediate response was obtained. The subsequent examination of the brain showed that the stimulation had been effective only when the needle had reached the white matter.

Similar experiments were made with hypertonic solutions and solutions of barium chloride, and the same results were obtained.

The author concludes that the grey matter is not irritable to chemical or osmotic stimulation. The stimulation which occurs after the application of certain substances to the surface of the brain is due to osmosis or diffusion taking place, which allows these substances to act on the white matter. The white matter is very irritable, and it responds very promptly to stimulation.

Goltz has shown that the cortex is devoid of irritability to mechanical injury. Further, electrical stimulation is, according to Loeb, only a form of chemical stimulation, the effect of the current being to produce a relative increase at the cathode in the concentration of the calcium precipitant ions. The general conclusion reached is therefore that the cortex is devoid of irritability to all kinds of stimulation, and that the response which is obtained on electrical stimulation of the cortex is due to spreading. W. CRAMER.

SOME THEORIES CONCERNING MUSCLE. E. FARQUHAR BUZZARD. (6) A Paper read before the Medical and Physical Society, St Thomas's Hospital, 1906.

ATTENTION is drawn to the tendency to look upon skeletal muscles as affording evidence in regard to the state of the nervous system rather than as highly specialised tissues having important functions of their own. Certain results of recent work on muscle physiology are examined, and an attempt is made to bring them into line with practical clinical medicine.

"Plain unstriated muscle is composed almost entirely of undifferentiated sarcoplasm; its contraction is of the slow type, and its excitability is of a low order, that is to say, it cannot be stimulated to contract by the short isolated induction shock, but will respond to the galvanic current, which is a more powerful stimulus of longer duration. Pale striated muscle is rich in fibrillar anisotropic substance, and relatively poor in sarcoplasm; its contraction is of the quick, sudden variety, and is easily elicited by isolated faradic shocks. Red striated muscle, such as is found in the toad, although containing both the fibrillar and sarcoplasmic elements, is relatively rich in the latter; its contraction and excitability occupy an intermediate position between those of the other two varieties." In man, no muscle is composed entirely of pale or entirely of red fibres; probably most of the muscles are made up of both kinds in varying proportions, which depend upon the kind of work for which the particular muscle is best adapted and most employed. Thus it is believed that sarcoplasm plays a great part in the production of tetanic contractions and in the maintenance of muscular tone; there is good reason to believe that red fibres are more numerous in those muscles which help to sustain the erect position of the human body, and that pale fibres predominate in those muscles which are concerned in quick, active movements.

Botazzi's theory of the "functional duality" of skeletal muscle is enunciated, viz. that "in striated muscle there are two contractile substances, the fibrillar and the sarcoplasmic, the former being characterised by its ready excitability, its quick contraction, and its poor resistance to various poisons, and the latter by its low excitability, its slow contraction, and its relatively great resistance to deleterious influences." This theory is applied to certain clinical facts:—

(1) *The Reaction of Degeneration.*—The skeletal muscle, which has been denervated, presents an early and marked diminution of its fibrillar substance, and a considerable development of sarcoplasm. This may explain, according to Ioteyko, the failure of the muscle to respond to faradic shocks, the persistence of response to the galvanic current, and the slow character of the contraction so

produced — quite characteristic of sarcoplasmic contractility. Even a small proportion of fibres retaining their fibrillar structure will be sufficient to yield a response to the faradic current; this is not uncommon in progressive muscular atrophy and in myopathy, and may explain the persistence of faradic excitability and absence of complete reaction of degeneration.

(2) *Thomsen's Disease, or Congenital Myotonia.*—It is impossible not to agree with Levi in thinking that the clinical features of this disease are identical with those of muscle in which there is an exalted state of sarcoplasmic activity.

(3) *Myoclonus.*—An exaltation of fibrillar excitability may account for the sudden sharp muscular contractions characteristic of this disease.

(4) *Myasthenia Gravis.*—It is worthy of consideration—although Buzzard does not urge this as a full explanation of all the facts—whether the paralytic phenomena may not be due to impairment or degradation of sarcoplasmic excitability, probably secondary to some more general influence of toxic, possibly of autotoxic origin. A. W. MACKINTOSH.

PATHOLOGY.

REGENERATION OF THE SPINAL CORD. (Recherches sur la régénérescence de la moelle.) G. MARINESCO et J. MINEA, *Nouv. Icon. de la Salpêtrière*, Sept.-Oct. 1906, p. 417.

(7)

IN this paper the authors describe the appearances found in the cords of dogs examined at periods varying from one to fifteen weeks after section, and also in two cases of compression of the spinal cord in man. Cajal's ammonia-alcohol and silver method was employed for the examination, and the conditions found were very similar in the experimental and in the pathological conditions.

New formation of nerve fibres occurred after the two fashions described both by the authors and by Cajal in peripheral nerves, viz. progressive growth of old fibres and multiplication by successive dichotomy of fibres of new formation.

Growth takes place from both upper and lower ends, but seems to be more prolific from the posterior roots upwards than from above downwards. The new fibres are of varying calibre; some are myelinated, but most are not, they end in cones of growth or spherical terminal masses, and frequently have fusiform swellings on their course.

Cells which they term "apotrophic" play the same important rôle here as they do in peripheral nerve regeneration. These are

fusiform cells which are found in the tissue between the divided ends a few days after section, with a fusiform nucleus, and rich in chromatin granules. Sometimes they are elongated, with several nuclei, and may be termed "protoplasmic bands." They have chemiotaxic properties, by which the young axons are attracted, and often actually drawn into their interior and nourished. The new nerve fibres extend by growing along these bands of apotrophic cells, and also by following the lines of the new vessels, forming a sort of perivascular reticulum. The course of the fibres is very irregular; many actually double back on themselves, and few succeed in crossing the cicatrix, so that in spite of the double cord and root regeneration, there is no functional restoration. To get this, it would be necessary for the proper neurons to become connected up as before, an event which can only occur with extreme rarity. J. H. HARVEY PIRIE.

(8) **CONTRIBUTION TO THE STUDY OF THE FINER ALTERATIONS OF NERVE FIBRES—PHENOMENA OF DE- AND REGENERATION—IN EXPERIMENTAL DEGENERATIVE PARENCHYMATOUS NEURITIS. (Contributo allo studio delle fini alterazioni della fibra nervosa—fenomeni de- e rigenerativi—nella neuriti parenchimatosa degenerativa sperimentali.)** MEDEA, *Riv. Sper. di Fren.*, Vol. xxxii., Fasc. 3-4.

DEGENERATIVE parenchymatous neuritis was produced in rabbits, guinea-pigs, and dogs by the injection of ether into nerves.

At the point of injection the nerve structures disappeared, and after a short time only remnants of myeline and axis-cylinder remained.

Very little change was seen in the central stump at the end of thirty days, but after about a hundred and eighty days much atrophy of the fibres was found to have taken place.

In the peripheral stump a true Wallerian degeneration followed the injection, but the author was unable to decide whether the degeneration of the myeline preceded that of the axis-cylinder or not.

The cells which occupied the sheath of Schwann after the removal of the myeline and axis-cylinder were derived from the cells of the sheath of Schwann by a process of rapid proliferation, and he suggests that they may have acted as phagocytes in the removal of the debris. No leucocytes were seen within the sheath of Schwann.

With regard to the regeneration of the nerve fibres, the author does not consider that the new fibres are derived from the proliferated cells of the sheath of Schwann, but he speaks of certain

fibrils which he has seen lying amongst these cells but not connected with them, in tissues treated by the method of Cajal, as the precursors of the new fibres. The origin of these is not stated.

He inclines to the view that regeneration occurs as an outgrowth from the axis-cylinder of the central stump. R. G. Rows.

ON THE PATHOGENESIS OF THE CORD CHANGES OCCURRING
(9) **IN THE COURSE OF POTT'S DISEASE. (Sur la pathogénie des altérations médullaires survenant au cours du mal de Pott.)** Italo Rossi, *Arch. de Neurol.*, Jan. 1906.

The first half of this paper gives an account of the different views that have been advanced as to the causation of the cord changes found in Pott's disease. Briefly these are: Purely mechanical osseous pressure; inflammatory myelitis from the compression of the cord by caseous material; anæmia from direct compression of the meningeal vessels and indirect compression of those of the cord; œdema from obliteration of the veins and lymphatics, both by compression and thrombosis; myelitis by direct extension from the meninges; myelitis, with or without meningitis, from the action of tubercle toxines. In the second half four cases are described, all varying in character, and the author concludes that the pathogenesis of the changes likewise varies in different cases; but when there is pachymeningitis, as in the great majority of cases, there are the necessary and sufficient conditions to produce mechanically circulatory disturbances (ischæmia and œdema), which are capable alone of explaining the changes. When the pachymeningitis is too slight to have this effect, it becomes necessary to invoke another factor, viz. the toxines from the tubercular foci, acting, perhaps, more through the collateral inflammatory œdema which they cause than directly on the nervous elements.

J. H. Harvey Pirie.

ON THE PATHOGENESIS OF THE SYMPTOMS OF BASEDOW'S
(10) **DISEASE. (Zur Pathogenese des Basedow'schen Symptomenkomplexes.)** Hirschfeld, *Centralbl. f. Nervenheilk. u. Psych.*, Nov. 1, 1906, p. 832.

The record is given of the case of a man, aged 36, who suffered from pains in the hip since November 1905. In March 1906 there was found to be an extensive and inoperable sarcoma of the pelvis. In the end of April there developed a dreamy mental state, and slight exophthalmus, becoming gradually more marked.

Later Graefe's and Moebius's symptoms appeared, also fine tremors in the fingers, and the pulse-rate reached 130 per minute. There was no palpable increase of the thyroid. These symptoms appeared and became extremely well marked in the course of a few days, and early in May the patient died.

The *sectio* discovered a sarcoma of the pelvis with numerous metastases. In the thyroid were three white tumours, the size of cherry stones. The healthy part of the gland showed none of the special changes described by Askanazy, etc., but here and there within the edge of and surrounded by the tumour lay isolated follicles full of colloid material. The sympathetic nerve showed no changes, and the only disease discoverable upon careful examination of the central nervous system was one minute secondary growth in the cerebral cortex.

The writer has been quite unable to find any record of another case of secondary growths in the thyroid gland accompanied by Basedow's disease, and the only ones in which primary thyroid tumour caused these symptoms were six collected by Ehrhardt in 1902. JOHN D. COMRIE.

MICRO-ORGANISMS IN ENDOCRANIAL OTITIC COMPLICATIONS. (Die Microorganismen bei den endokraniellen otogenen Komplikationen.) (11) Dr HASSLAUER, *Centralblatt f. Ohrenheilkunde*, Bd. 5, H. 1 and 2.

ALTHOUGH the bacteriology of middle ear suppurations has been thoroughly investigated, our knowledge of the organismal cause of resulting endocranial complications is exceedingly scanty. Infections of the lateral sinus are usually due to streptococcic invasion, but staphylococcus aureus and albus, pneumococcus, B. coli, B. pyocyaneus, and other organisms have been found. Extradural abscess is most frequently produced by the pneumococcus. The data are insufficient in cases of subdural abscess to come to any conclusion. In brain abscesses, in acute cases, the streptococcus is most frequently found, and next to it the diplococcus: in chronic cases several organisms are usually present. The cases of meningitis where the actual pus was examined are too few in number to allow of any conclusion being drawn, but the cerebro-spinal fluid obtained by lumbar puncture has been examined in a large series of cases. In these diplococci, and next to them streptococci, are most frequently found; sometimes anærobic bacilli are met with. In many cases the fluid is stated to have been sterile. Brieger has suggested as a possible explanation that these cases are due to toxins, or else that the organisms are very attenuated, therefore many authors lay more stress on

immediate microscopic examination than on the study of cultures. It is generally agreed that the presence of bacteria must be demonstrated before a diagnosis of either purulent or tuberculous meningitis can be made; but even here error may creep in—where a cerebral abscess has burst; while, on the other hand, the examination of the fluid may be negative, although a purulent meningitis is present. The diagnostic value of lumbar puncture has been very variously estimated, some authors placing the greatest trust in it, others being entirely sceptical.

The same organisms, then, which cause middle ear suppurations, also bring about their endocranial complications, but that the various complications are induced only by certain organisms does not appear to be the case from what is stated above; other factors, such as the virulence of the organism and the resisting power of the individual, must also be considered.

W. G. PORTER.

A CONTRIBUTION TO THE PATHOLOGY OF CHOREA.

(12) POYNTON and HOLMES, *Lancet*, October 13, 1906, p. 982.

THIS paper is divided into four parts.

Part I.—Introductory. The authors believe that the great majority of cases of chorea are rheumatic in nature, and hold that the disease is a direct result and not a complication of rheumatism. Acute rheumatism is the result of an infection by the "diplococcus rheumaticus," a conclusion borne out both by clinical and bacteriological evidence. Acute arthritic rheumatism has been produced in animals by experimental inoculation with a diplococcus isolated from the cerebro-spinal fluid and inflammatory exudations obtained from a case of acute rheumatism and chorea (Wassermann, 1899). In 1900, Poynton and Paine confirmed this observation, and also demonstrated the diplococcus in the pia and brain in a fatal case of chorea, and they produced choreiform movements in the rabbit by inoculating it with the organism, a result also observed by Meyer and by Beattie.

Part II.—The Investigation. In two fatal cases of chorea of rheumatic origin, and also in a case of "chorea gravidarum," they were successful in finding the diplococcus. From the heart blood of the first case, a girl of seven, they obtained a pure culture of the organism, which was also demonstrated in the pericardium and in the pia and brain, whilst experimental inoculation produced multiple arthritis and pericarditis in rabbits. The microscopical changes in the brain are described, the most important of these being congestion and occasional thrombosis of the vessels, especially the smaller arteries of the membranes and brain; cellular infiltra-

tion around these; small areas of necrotic softening; chromatolysis and other degenerative changes in the nerve cells, etc. The second case, that of a boy of fourteen, gave practically identical results; whilst in the third case, that of a pregnant female aged twenty-one, with no previous history of rheumatism or chorea, and who developed a very severe attack of chorea at the second month, the autopsy revealed recent vegetations on the mitral valve; and on microscopical examination of the brain, changes similar to those described above were found, the vascular changes being less, and the degenerative changes in the cells being much greater, than in the other two cases.

Part III.—The Pathology of Chorea. In this section the authors discuss the nature of the disease and its relationship to other manifestations of rheumatism, their conclusion being that the causation of chorea is to be found in the action of bacterial toxins on the brain, producing vascular, inflammatory, and degenerative changes, though, whether these are due to direct local invasion or to a diffusion of toxins, they are not yet prepared to say.

Part IV.—Chorea and Pregnancy. All the clinical points in this (the third) case, as in numerous other recorded cases, seem to show that the chorea of pregnancy is of the same nature as the chorea of childhood, and that it is a manifestation of acute rheumatism during a critical physiological period of special strain.

These cases afford additional evidence that the "diplococcus rheumaticus" is the infective agent in acute rheumatism.

W. E. CARNEGIE DICKSON.

THE PATHOLOGICAL ANATOMY OF EPILEPSY. (Sull' anatomia patologia dell' epilessia.) (13) GUIDO SALA, *Rivista Speriment. di Freniatria*, 1906, Vol. xxxii., p. 488.

THE first thirteen pages of this article are taken up by a detailed review of the literature and a discussion of the work done as supporting one or the other of the two rival sets of findings: those in the cortex and those in Ammon's horn. An account is then given of ten cases examined microscopically. The staining methods employed were mostly: for the blood-vessels, hæmalum, hæmatoxylin; for the neuroglia, Weigert's, Benda's, and Dott's modification of Heidenhain's; for the nerve cell, Nissl's and Pappenheim's. The cases were all ones of essential epilepsy, not symptomatic.

The results reached were as follows:—

(1) In all the cases the cortex shewed changes more or less grave in relation with the duration and severity of the malady;

these changes concerned the vessels, glia, and nerve cells; they varied closely with the degree of dementia, being most marked in a case of idiocy.

(2) The changes were most marked in the ascending frontal and ascending parietal areas; they were found, however, elsewhere in the cerebral cortex, particularly in the frontal and occipital lobes. Ammon's horn, especially the convoluting grey layer, was also implicated, but always to a less extent. In one case, with idiocy, there were hypertrophied spider cells here, with long fibrillar prolongations that had intimate connection with neighbouring nerve cells.

(3) The vascular changes were: dilatation and atrophy of the wall, dilatation of the perivascular lymphatic sheath, which was filled with a leucocytic infiltration; disseminated punctiform hæmorrhages and larger extravasations occurred in cases that succumbed during any illness or epileptic attack. No case shewed the characteristic changes due to plasma cell accumulation, such as is seen in paretic dementia.

(4) The neuroglia changes related especially to the sub-pial layer. A thick feltwork of fibrillæ was formed, in the midst of which were found glia cells of average size. In the external layer of the cortex also there occurred a large number of glia cells, the robust prolongations of which reached, some to the vessel walls, others to the sub-pial feltwork, contributing to the formation of this. There were a fair number of monster glia cells also present. In the cases with dementia, evidences of active proliferation of the glia cells were seen.

In Ammon's horn the special feature was the number of monster cells.

(5) The changes in the nerve cells were extremely inconstant, varying from slight chromatolysis to advanced cellular degeneration. They are attributable less to the disease than to the mode of death, and similar findings may be seen in old age and many other conditions. The gigantic cells described by many authors, and attributed by them either to a functional hypertrophy in connection with the epileptic attacks or to a teratological condition, were found chiefly in the Rolandic region. The author disagrees with those who attribute significance to their presence, and thinks that they are probably merely large Betz cells. He thinks that not enough attention has been paid to the variation in size these may present in the normal brain.

(6) In two cases a slight chronic lepto-meningitis was found; this was present chiefly over the Rolandic region, and was in relation to the more marked glia growth in the underlying cortex of that area.

(7) In conclusion, the author is of opinion that none of the

changes described are at all characteristic or specific of epilepsy, but may be found in many other morbid conditions, and even in simple senility. It is probable that they are all secondary to the circulatory disturbances mechanically produced by the attacks. In the case of epileptic idiocy, however, no doubt some of the changes, particularly the glia overgrowth, may be ascribed to defect in development. ERNEST JONES.

NEW RESEARCHES ON THE NATURE OF THE TOXIC PRINCIPLE CONTAINED IN THE BLOOD SERUM OF EPILEPTICS. (Nuove ricerche sulla natura dei principi tossici contenuti nel siero di sangue degli epilettici.) CARLO CENI, *Rivist. Speriment. de Freniat.*, Oct. 1906, Vol. xxxii., p. 451.

(14)

THE latest results of the author's well-known researches on this subject may be summarised as follows:—

Animals immunised with epileptic blood serum that is hypotoxic for man suffer no inconvenience. Animals submitted to a process of immunisation with the epileptic blood serum that is hypertoxic for man die rapidly if they are injected at once with half the dose necessary to protect an animal against the hypotoxic serum. The intoxication, however, presents nothing in common with the phenomena of epilepsy; the animals suffer from a grave hæmolytic process similar to that produced in man by injection of the same serum. The biologic reactions of the hypotoxic serum are in no sense specific. Their precipitating properties are constantly identical with those of normal blood serum. The hypertoxic serum, as a rule, behaves exactly in these respects as the hypotoxic one. In a few cases, however, it shewed a specific precipitation reaction in the presence of an antiserum prepared from the same serum. ERNEST JONES.

CLINICAL NEUROLOGY.

A CASE OF DIFFUSE SUB-ACUTE POLIOMYELITIS IN INFANCY. (Étude d'un cas de poliomyélite diffuse subaigue de la première enfance.) P. ARMAND-DELILLE et G. BOUDET, *Nouv. Icon. de la Salpêtrière*, Sept.-Oct. 1906, p. 441.

(15)

THIS case is remarkable as commencing in a child of two months, with death at six months. Weakness was first noticed in muscles of neck, then the extremities and trunk were progressively involved.

Sensibility, as far as could be judged, remained quite intact. The organic functions were unaffected; there was never any rise of temperature; the cerebro-spinal fluid showed a slight lymphocytosis. In the cord the changes were confined to the anterior cornua—a simple atrophy of practically all the motor cells, with some neuroglial proliferation, but no vascular changes. The anterior roots and motor nerves were degenerated, while the muscles were extremely atrophied, with all the characters of a simple myelopathic atrophy. A short abstract is given of the few cases of this disease which have been recorded in children—none hitherto under one year. J. H. HARVEY PIRIE.

(16) **LUMBAR PUNCTURE AND CYTOLOGICAL DIAGNOSIS — IMPORTANCE OF DIAGNOSIS. (Ponction lombaire, etc.)** L. LARUELLE (de Liege), *Journ. de Neurol.*, Nov. 20, 1906, p. 576.

DR LARUELLE draws attention to the importance of the enumeration of cellular elements in the cytological diagnosis of cerebro-spinal fluid.

He uses the counting cell, first used by Fuchs and Rosenthal, preferring that to the method of Widal, as being more practical and exact and requiring a shorter time for its performance.

His method, requiring only microscope, mixing pipette, and counting cell, avoids the necessity of centrifuging and the various manipulations necessary in fixing and preparing films. He emphasises the fact that, in the grosser forms of meningeal affections there is no difficulty in arriving at a conclusion from the mere appearance of the fluid, and that it is only in the narrow margin between slight meningeal affections, with a slight leucocytosis, and normal cerebro-spinal fluid containing a few cells, that any difficulty of diagnosis arises.

Dr Laruelle lays down the proposition, as a result of experiment and more recently of a series of fifty cases, of which six were normal, that "there is a pathological reaction present when the enumeration, repeated two or three times on a fresh liquid, gives an average of more than five leucocytic elements per cubic millimetre." He gives two interesting examples of very early diagnosis by this means, where the other methods of examination of the fluid gave negative results, and in which surgical interference proved the correctness of the diagnosis, and enabled an earlier operation being performed than otherwise would have been possible.

Dr Laruelle points out, in conclusion, that the amount of cerebro-spinal fluid required is less than in the other method,

a point sometimes of importance, and that the comparison of results by different observers is of much greater value, as regards the simplifying of the technique. DUNCAN LORIMER.

THE FUNCTIONS OF THE CEREBELLUM AND THE SYMPTOMS OF ITS DISEASE. (17) C. L. DANA (of New York), *N.Y. Med. Journ.*, Oct. 6, 1906.

THE function of the cerebellum is essentially reflex and automatic; its activities are never attended by consciousness. Symptoms of cerebellar disease are conveniently divided into three groups—(*a*) those due to direct injury or irritation of the cerebellum, (*b*) those due to increased intracranial pressure causing general cerebral or local pontine or medullary symptoms, and (*c*) those due to sudden vascular changes or irritations leading to seizures.

Of the individual symptoms, the most important are vertigo, nystagmus, constrained positions, muscular weakness, and reeling gait. The author refers to the symptoms produced by small cerebellar hæmorrhages or softenings as forming the most typical and distinctive group of cerebellar symptoms.

C. H. HOLMES.

ON SEROUS MENINGITIS. (**Ueber seröse Meningitis.**) RIEBOLD, (18) *D. med. Wchnschr.*, Nov. 15, 1906, p. 1859.

THE writer gives a brief historical account of this term used as long ago as 1844 by Barthen, and by Billroth in 1869, to describe an acute non-tubercular meningitis. It was only in 1887, however, that Eichhorst clearly demonstrated the fact that cases arise acutely and remain serous, *i.e.* non-purulent, throughout their whole course. In 1893, Quincke, as the result of lumbar punctures, gave an exact description of the clinical picture.

The present paper contains details of eight cases in which the symptoms generally speaking were headache, pains in the body and limbs, high temperature, slow pulse, unconsciousness, inequality of the pupils, and often loss of light reflex, vomiting, frequently ocular and other paralyses, usually leucocytosis in the blood. The most important sign is given by the spinal fluid, which is clear, of high pressure (200 or even 300 mm. of mercury), contains only a few lymphocytes, gives the albumin reactions, and is sterile as tested by making cultures or animal injections. Six of these cases recovered after an illness of some weeks.

The writer agrees with Quincke that a case of serous meningitis may often be the exacerbation of a compensated chronic hydrocephalus which is present as the result of an earlier meningitis. He believes also that so far from being a rare affection, serous meningitis in slight degree is a very common affection, particularly as a complication of acute infectious diseases. He further points out that the serious cases are not to be distinguished from tubercular and purulent meningitis save by examination of the spinal fluid. JOHN D. COMRIE.

CHRONIC DIPHTHERITIC BULBAR PARALYSIS. (Paralysie bulbaire chronique et progressive, d'origine diphtérique.)
(19) TINEL and SIREDEY, *Bull. et Mém. de la Soc. Méd. des Hôp. de Paris*, Nov. 22, 1906, p. 1126. SCHERB, *ibid.*, Dec. 13, p. 1225.

THE extreme rarity of chronic progressive bulbar paralysis due to diphtheria is illustrated by the fact that the present case is only the eighth on record (*v. Review of Neurol. and Psychiat.*, 1904, pp. 461 and 562).

A sailor, aged 24, was admitted to hospital with a history of deaf-mutism that had suddenly developed a month ago. This condition, which was found to be of hysterical origin, and to have been brought on by a debauch, yielded in a fortnight to appropriate treatment. Definite organic lesions, however, remained, which were as follows: atrophy of the tongue, with fibrillar contractions; paralysis of the larynx, with atrophy of the vocal cords; slight paresis of the lips and diminution of the senses of taste and hearing. Laryngoscopic examination showed complete paralysis of the dilators and of the crico-thyroid; the paralysis of the constrictors was less marked. The voice was hoarse. The deep reflexes were normal, the general health was good, and sensation seemed everywhere intact. There was neither sugar nor albumin in the urine.

The onset of these symptoms was traced to a severe attack of faucial and laryngeal diphtheria, requiring intubation, that had occurred in 1895, and was followed by paralysis of the palate and larynx. The patient did not receive antitoxin and had to wear the intubation tube for sixteen weeks. The palatal palsy lasted until 1901 and then gradually disappeared without leaving any trace. The paralysis of the larynx, on the other hand, became more marked. Even at the onset the voice was affected, and there were occasional suffocative attacks. Four years after his attack of diphtheria, the patient was under Lermoyez, when the mere rapid ascent of a staircase was enough to bring on the attacks, and the stridor was so loud at night that he had to be put in a

room by himself. At that time the constrictors of the glottis were less affected. The voice was noted as good and not husky. Atrophy of the tongue was not present then, and was not noticed till June 1905.

After travelling from the larynx along the nerve-trunks, the diphtheritic toxines probably affected the laryngeal centres in the medulla at the onset of the disease, and the contiguous nuclei subsequently became involved by a process of sclerosis or atrophy.

J. D. ROLLESTON.

A CASE OF EXTENSIVE THROMBOSIS OF THE CEREBRAL SINUSES. (Ein Fall von ausgedehnter Thrombosierung der Hirnsinus.) (20) WIMMER, *Berlin. klin. Wchnschr.*, Nov. 12, 1906.

THIS is the record of a case of extensive and apparently non-infective thrombosis of the cerebral veins; the writer also refers to two other very similar cases.

A man of 52 was suddenly seized, at work, with paralysis of the right arm, and general trembling followed by weakness in the right leg, and the same evening by twitching in the right side of the face. There was, however, during the first day no loss of consciousness. Convulsive attacks beginning in the right arm, followed by a condition of coma, lasting half an hour or thereabout, developed and recurred at frequent intervals, the patient dying at the end of four days.

On account of the sudden onset, the Jacksonian symptoms, and marked signs of high pulse tension and diseased arteries, the case was regarded as a cortical arterial apoplexy. The *sectio*, however, showed thrombosis of all the large sinuses, and of the minute pial veins in many places with small hæmorrhages. The cerebral arteries were quite unaffected. JOHN D. COMRIE.

CHOREA GRAVIDARUM. HERBERT FRENCH and H. T. HICKS, (21) *Pract.*, Aug. 1906.

OF twenty-nine consecutive cases of this disease treated in Guy's Hospital, only three died. Barnes gave the mortality as 43 per cent. and Buist as 20 per cent., but these figures were founded on cases taken from different sources in literature, and would probably not include mild cases with a favourable and uninteresting course. The majority of the patients were about twenty; only three were as old as thirty. Eighteen were primiparous. A distinct tendency to recurrence in later pregnancies was noted. In none of the cases that recovered did the temperature rise above

99°; in all the fatal cases there was marked pyrexia. If there is no obvious complicating cause, a rise of temperature above 100° is of grave prognostic import. The severity of the choreic movements is of little prognostic value. Treatment is similar to that employed in chorea without pregnancy. Induction of labour is rarely indicated, for, on the one hand, the severest cases get better without it, and, on the other, it is too late to induce labour when pyrexia has set in. Moreover, induction of labour is not a specific cure for the condition. HENRY J. DUNBAR.

(22) **ON THE SIGNIFICANCE AND FREQUENCY OF THE UNILATERAL MANIFESTATIONS OF IDIOPATHIC EPILEPSY. (Sopra il significato e la frequenza delle manifestazioni emilaterali nell' epilessia essenziale.)** CARLO BESTA, *Rivista Speriment. di Freniats.*, Oct. 1906, Vol. xxxii., p. 665.

THIS subject is dealt with fully in a monograph of over sixty pages. Forty-three cases are described in detail, and are arranged in the following groups:—

1. Fourteen cases of Idiopathic Epilepsy. These were absolutely typical cases except for one respect, the unilaterality of the manifestations. In three instances these were confined to one side throughout, and there was conjugate deviation of the eyes to that side; in six instances the tonic phase was confined to one side, but the clonic movements were general: in nine instances the manifestations were bilateral throughout, but more marked on one side: in some of the latter cases the manifestations were bilateral throughout in some attacks, but only in the clonic stage in others. In all these cases there was a weakness on the corresponding side after the attacks; in ten of the cases the weakness persisted in the face during the inter-paroxysmal periods; in seven the knee-jerk after the attack was increased on the one side; and in eight Babinski's extensor response was present on that side only.

2. Five cases of Unilateral Attacks, associated with signs of an old cerebral lesion. These correspond to Freud's "cerebral paralysis without paralysis," as there was no weakness, only signs such as Babinski's, etc. These were due to a slight infantile hemiplegia of which the epilepsy was left as almost the only trace. Some cases were extremely difficult to differentiate from those in the first group, there being no sharp dividing line.

3. Twenty-one cases of Bilateral Epilepsy. In the inter-paroxysmal periods it was found that of these, nine had unequal facial innervation on the two sides, four had deviation of the tongue, three asymmetry of the knee-jerks, nine asymmetry of the plantar reflexes, Babinski's sign being present in one. In six of

the cases the movements were far more pronounced on one side, always the same one, and there was conjugate deviation of the eyes towards that side. The post-paroxysmal phenomena included increase of the facial weakness in those suffering therefrom, asymmetry of the knee-jerks in thirteen cases, presence of Babinski's sign in three cases; in two of the latter it was unilateral. The author concludes this section by demonstrating the impossibility of sharply dividing this group from the first. He considers it proved that the diminished cortical resistance in epilepsy affects one hemisphere more than the other in a large number of cases.

4. Three cases of Traumatic Epilepsy. In one of these the manifestations were bilateral throughout, in another loss of consciousness was the first sign, and was followed by unilateral convulsions, thus exactly resembling the cases in group one.

Many authors are quoted in confirmation of the above results. Especially striking were those of Redlich, who in 150 cases found asymmetry of the reflexes after the attack in over 40 per cent. The bearing of the observations on the pathology of epilepsy is fully discussed. The author favours the position taken by Marie, Féré, and others, who maintain the probable existence of an anatomical basis for the disease, in opposition to Binswanger, who claims that all the pathological changes described are secondary to the functional disturbance. The author, however, agrees with Binswanger that all the changes hitherto described are to be explained thus, but anticipates more exact results with the aid of finer methods of research. He does not discuss the possibility of the unilateral changes he describes being due also to functional disturbance.

The final conclusions are summed up as follows:—

1. In the majority of cases of epilepsy there exist indubitable signs of functional asymmetry in the interparoxysmal period. These concern the muscular strength, and the deep and superficial reflexes.

2. In many cases this asymmetry corresponds with the form of convulsion, as noted above under group 1. There is no constant relation, however, between the intensity of the asymmetrical signs and the variety of convulsion described in that group. The unilateral signs in the fit affect always the same side.

3. The fit, of whatever variety, always has the effect of accentuating the above signs for a short while, and sometimes elicit them when not otherwise present.

The initial cry is observed only in cases—though not in all of these—in which the functional asymmetry affects the right side.

ERNEST JONES.

A CONTRIBUTION TO THE SYMPTOMATOLOGY OF PETIT MAL. (Beitrag zur Symptomatologie des Petit Mal.) (23) BRESLER (of Lublinitz), *Psych.-Neur. Wchnschr.*, Aug. 18, 1906.

AN attack of petit mal, as a rule, interrupts the occupation of the moment, which is resumed when the attack passes off. Bresler endeavoured to ascertain, in a patient with extremely numerous attacks, how far it was possible to continue some definite activity throughout the attack. The patient was told to count from one upwards, and in some of the attacks he continued to count throughout, in others he merely counted one or two additional numbers. The author found that after the attack the patient was sometimes able to name objects shown during the attack.

C. MACFIE CAMPBELL.

CONTRIBUTION TO THE KNOWLEDGE OF STATUS HEMIEPILEPTICUS. (Beitrag zur Lehre von Status hemiepilepticus.) (24) BERNHARDT, *Berl. klin. Wchnschr.*, Nov. 5, 1906, p. 1443.

THE writer records the case of a woman, aged 35, in whom epileptic seizures affecting especially the left side of the body appeared. The first attack came on in the night and was followed by some weakness of the left side. The following afternoon there was a typical epileptic fit, and another on the day succeeding. Afterwards, loss of consciousness and almost continual spasms on the left half of the body persisted, there was conjugate deviation of head and eyes to the left, and incontinence of urine and fæces. Severe general fits finally appeared every half-hour. The right side of the skull was trephined, but no abscess, tumour, cyst, or other abnormality could be found, and two hours after the operation the patient died. No further change was found post-mortem.

The writer gives a précis and bibliography of similar cases, some of which recovered. He refers to the difficulty of distinguishing such cases from cerebral tumours, but mentions that mere trephining is sometimes remedial.

JOHN D. COMRIE.

THE RELATION OF BROMIDE ACTION TO THE METABOLISM OF EPILEPTICS. (Die Beziehungen der Bromwirkung zum Stoffwechsel der Epileptiker.) (25) J. HOPPE, *Neurolog. Centralbl.*, Nov. 1, 1906, S. 993.

THIS paper is an exposition of the principles underlying the Toulouse-Richet treatment. The author first refers to Landen-

heimer's researches, which shewed that no therapeutic results followed bromide administration until an equilibrium is established between its intake and excretion; this occurs only after the body is saturated by the accumulation of a certain amount of bromide, for which the giving of thirty grams is necessary. These researches have since, however, been supplemented. Although their principle has been established, it has been shewn that Landenheimer over-estimated the percentage of bromide in Bromuatrium, and also failed to take into account the varying excretion capacity in different people. This latter point is the explanation why children tolerate bromide better than adults, because their kidneys are less damaged. The other important factor is the amount of salt taken in the food. No therapeutic results are observed until one-third of the chlorine of the blood serum has been replaced by a chemically equivalent amount of bromine. It is known that bromide accumulates in the vessels, chiefly in the blood serum, and not in the organs of the body. When more than this amount of chlorine is displaced, intoxication is liable to occur. To determine when this point has been reached, one may examine the blood serum, 25 c.c. being necessary, or, more conveniently, the gastric juice, for it has been shown that the same rule applies here also. Now, clearly, when less chloride is taken in the food, this saturation point is reached sooner. For instance, it takes three to four weeks to reach it on an unrestricted diet, but only three to four days on a Toulouse-Richet salt-free diet. It follows that in the former case the administration of even four to six grains of bromide daily may go on for years without toxic symptoms, whereas in the latter they may appear after some weeks and quite suddenly. When this occurs the proportion of chlorine to bromine in the gastric juice may be only 0·09 to 0·26. This danger can be avoided by careful procedure and by the gradual introduction of the salt-free diet. Sudden changes in diet or in the variety of bromide employed bring about a variation in the chlorine-bromide proportion. Again, the effect of various diseases, especially the acute specific fevers, has to be noticed. In many of them, particularly in croupous pneumonia, salt-excretion is diminished, with the result that bromide retention occurs. Hence the lessened frequency of epileptic attacks during fevers, and also the increased risk to life owing to the depressant effect of the bromide. In other infections, such as a streptococcic, the reverse obtains.

ERNEST JONES.

THE QUESTION OF PROTOPATHIC AND EPICRITIC SENSIBILITY AND THE DISTRIBUTION OF THE TRIGEMINUS NERVE (3rd branch). C. L. DANA (of New York), *Journ. of Nerv. and Ment. Dis.*, Sept. 1906.

(26)

THE monograph (*Brain*, 1905) of Head, Rivers, and Sherren contends that the afferent nervous system is composed of three different systems which conduct different forms of sensibility to the brain. (1) A protopathic system composed of fibres running in connection with the sympathetic and the vessels which furnish a low degree of sensibility to the skin and viscera. (2) A protopathic system composed of fibres running in the motor nerves which supply the sense of movement, of deep pressure, and appreciation of position, of muscles, joints, and tendons. By the protopathic system, pain, temperature, localisation, and position are appreciated, but not to a very delicate extent. (3) An epicritic system which supplies the skin alone and enables us to appreciate light touch, localisation, and minor degrees of temperature, ranging between 22° and 40° C.

If this scheme were true for the trigeminal nerve, then one would expect that the surgical division of the nerve would leave at least the second form of protopathic sensibility, *i.e.* appreciation of movement, position, of deep pressure, and perhaps of extremes of temperature. Dana contends that while such an arrangement may or may not apply to the spinal nerves, it is certain that the central nervous system and the cranial nerves conduct sensation in a different way. To support the assertion, he cites actual cases, giving the detailed history of two. In the first case reported there was complete section of the inferior branch of the trigeminal nerve; every form of sensibility which it is possible to test was lost over the area supplied by this branch. In a complete paralysis of the seventh nerve there was no loss of deep sensibility or sense of position, although according to the scheme, section of the motor nerve should eliminate the second protopathic system. In the second case reported, one of hysteria major with anæsthesia of the trigeminus, there was absolute loss of all forms of sensibility, both deep and superficial; if there is a differentiation of sensibility into two types, it is in just such cases that one would expect to find, although not necessarily, dissociation of the two modalities.

In cases where the pons and medulla are involved and striking dissociation of sensibility may occur (*i.e.* temperature, pain, and touch differently appreciated), the loss of the temperature sense may be absolute. From such cases Dana concludes that, at least in the central nervous system, temperature sensations, for example, are conducted by paths which conduct all degrees of sensation. The changes in the sensibility observed by the author in cases of

cerebral hæmorrhage involving the posterior segment of the internal capsule could not be explained by the scheme referred to.

C. H. HOLMES.

ON VOLUNTARY WIDENING OF THE PUPILS. (Ueber willkürliche Erweiterung der Pupillen.) (27) BLOCK, *Deut. med. Wchnschr.*, Nov. 1, 1906, p. 1777.

THE writer records the case of a man who is said to possess the power of voluntarily widening his pupils by mental concentration on the subject. Two references are given (*Neurolog. Zentralbl.*, 1899, Nos. 1 and 11; and *Deut. Zeitschr. f. Nervenheilk.*, 1895, No. 2) to previously recorded cases of a similar nature. In the present case the patient, a morphinist, gazes into vacancy and wills that his pupils should widen, whereupon after three or four seconds they dilate to about one-fourth more of their previous width. At the same time the face reddens. He cannot voluntarily narrow them, and the power to maintain the increased width lasts only about a quarter of a minute.

JOHN D. COMRIE.

THERE IS NOT SUCH A THING AS HYSTERICAL MYDRIASIS. (La mydriase hysterique n'existe pas.) (28) CH. SAUVINEAU, *Rev. Neurolog.*, Nov. 30, 1906.

THE author endeavours to show in this paper that functional affections of the pupil, especially dilatation, do not occur. Unless precautions are taken the diagnosis of hysteria may be made, though the subsequent course of events will reveal the true nature of the case.

He records three cases where the question of hysteria arose, all of which, however, proved eventually to be organic lesions.

Case 1. Sudden loss of sight in the right eye, the right pupil being slightly dilated and immobile. Consensual pupil reaction to light present when the left eye was illuminated, absent for the right eye. The only objective sign was a very slight pallor of the right disc. There was a history of a similar occurrence about three weeks previously, with a sudden return of vision.

Diagnosis of a vascular lesion, probably hæmorrhage into the optic nerve, was made.

Four days later, patient regained sight in the right eye, and direct light reflex returned. There was still some concentric reduction of the right visual field. A month later the condition had relapsed with similar pupil changes, and eventually optic atrophy developed completely.

Case 2. Complete blindness followed a traumatic injury to the eye. At a large hospital the diagnosis of hysterical amaurosis was made. On the second day was seen by the author. Nil abnormal in the eye or media except dilatation of pupil on the affected side and loss of direct light reflex, though the consensual reflex still remained in that eye. Prognosis of rapid atrophy of the optic nerve was made, and actually occurred.

Case 3 was that of a woman who fell in the street after a fright from a motor car, and bruised her temporal region very severely. Exactly the same eye conditions resulted as in the first two cases and again hysteria was diagnosed by the medical attendant, but optic atrophy resulted.

The author regards the eye condition, *i.e.* blindness, with dilatation and loss of the light reflex, as characteristic of organic disease, though a secondary atrophy does not appear for several weeks.

The condition is not a true paralytic dilatation in which the pupil is immobile, though the perception of light remains and the consensual reflex from the healthy eye produces no contraction, such cases being met with in third nerve paralysis.

In the third nerve cases the centrifugal fibres of the optic nerve are affected—in the cases described the centripetal fibres.

In hysteria neither path is interfered with and so the reflex remains.

The author utters a word of warning against deception by such drugs as atropine. C. M. HINDS HOWELL.

A CASE OF TOTAL AND COMPLETE UNILATERAL OPHTHAL-
(29) **MOPLEGIA, WITH BLINDNESS OF THE SAME SIDE. (Un cas d'ophtalmoplégie unilatérale, totale et complète, avec cécité du même côté.)** BOUCHAUD, *Journ. de Neurol.*, Nov. 5, 1906, p. 549.

THIS is one of the rarest forms of ophthalmoplegia. The patient was a man aged 40, a worker in lead, with no definite history of syphilis. The first symptom was internal squint of the right eye, and the full development of the ophthalmoplegia occupied over two months, during which period there were two attacks of unconsciousness, not followed by paralysis, and headaches of increasing severity in the occipital region and on the right side of the head. All the muscles of the right eye were paralysed, and there was loss of reaction to light and accommodation, complete loss of sight and ptosis. At first the appearance of the fundus was normal, but later there developed well-marked optic atrophy. There was also loss of the sense of smell on the right side. Under treatment with large doses of iodide and mercury, recovery was complete as

far as the movements of the eye were concerned, but the blindness persisted. The lesion was probably a syphilitic meningitis situated in the anterior and internal part of the middle cranial fossa in the region of the cavernous sinus. HENRY J. DUNBAR.

THE MEANING AND LOCALISATION OF REFLEX PUPILLARY
(30) **PARALYSIS. (Begriff und Lokalisation der reflectorischen Pupillenstarre.)** L. BACH (Marburg), *Die ophthalmologische Klinik*, June 22, 1906.

THIS article commences by pointing out the necessity for accurately differentiating between a reflex paralysis of pupils, an amaurotic paralysis, and an absolute paralysis. The author defines a reflex paralysis as one in which the pupils show no reaction directly or indirectly to light stimuli, to reflex nervous stimuli, nor to psychical stimuli, but react promptly to convergence. Such a pupil is narrow, its width usually lying between 1·5 and 2·5 mm. He points out that it is distinguished from amaurotic paralysis by the difference in the width of the pupils and by the reaction to nerve stimuli and psychical stimuli, though in certain diseases the one passes over into the other and makes diagnosis impossible.

The differentiation from a "partial" absolute paralysis (*i.e.* a general paresis) is fairly easy, since in this case all the movements will be more sluggish, the convergence reaction included, and the width of the pupils will be greater.

The author, after reviewing various hypotheses as to the site of the pathological lesion or lesions leading to reflex pupillary paralysis, puts forward his own views as to the probable existence of an inhibitory centre for the light reflex and for pupillary dilatation in the distal portion of the medulla oblongata. While he admits that he can give no strict proof of the existence of such a centre, he thinks that a centre in such a position will best correlate the known facts on the subject.

LESLIE PATON.

ANATOMICAL PROOF OF THE VALUE OF THE PARADOXI-
(31) **CAL REFLEX. (Preuve anatomique de la valeur du reflexe paradoxal.)** ALFRED GORDON, *Rev. Neurolog.*, Nov. 30, 1906.

SINCE his first communication on this subject in the *Revue Neurologique* of November 15, 1904, the author has made a further series of observations, all of which tend to confirm his original conclusions. The cases he records in this communication exhibit the relation of the paradoxical reflex to cerebral compression, and further admit of definite proof in this respect from an anatomical point of view.

He quotes two cases in full, in the first of which there was evidence of pressure on the right cerebral cortex, with presence of an exaggerated knee-jerk on the left side, and the presence of the paradoxical reflex. Babinski's sign, Oppenheim's sign and ankle clonus were, however, all absent. Operation was performed and the pressure relieved with disappearance of the reflex in question, which again appeared on recurrence of pressure.

The second case exhibited an exactly similar association of symptoms, including the reflex due to traumatic injury to skull. Operation was successful and the reflex disappeared. The author concludes that the paradoxic reflex is an early and valuable sign of irritation or destruction of the pyramidal system and may appear before Babinski's sign. C. M. HINDS HOWELL.

BONE REFLEXES. (Réflexes osseux.) D. NOICA and L. STROMINGER, (32) *Rev. Neurol.*, November 1906.

THERE is very little literature on this subject. In 1901, Déjerine made mention of such reflexes; in 1903, Von Bechterew described contractions of certain muscles on percussion of certain bony points, which he regarded as periosteal reflexes; and in 1905, Bertolotti and Valobra published a study of bone reflexes in the lower limbs. In 1906, Strumpell drew attention to similar phenomena in both upper and lower extremities, which he considered to be aponeurotic and periosteal reflexes.

The authors have carried out further investigations. They use a light percussion hammer for the arms, and a much heavier one for the legs. They find it necessary to strike gently, always using the same force; heavy blows excite general contractions, from which it is impossible to pick out the one required.

Upper Extremity.—The arm is slightly flexed and pronated, the wrist slightly extended. Percussion on the styloid process of the radius causes contraction of biceps and supinator longus; on the styloid process of the ulna it causes contraction of the epitrochlear muscles. Percussion on the epicondyle causes contraction of the deltoid, on the olecranon it causes contraction of the triceps.

Lower Extremity.—(1) Dorsal decubitus, legs flexed on thighs, heels on the bed, knees slightly separated. Percussion on internal tuberosity of tibia causes adduction of knees. On the tuberosity or internal malleolus it causes contraction of the inner hamstring. (2) Lateral decubitus, leg flexed, tendo Achillis stretched, percussion on internal malleolus causes contraction of sural muscles; on the iliac crest it causes contraction of hamstrings.

The authors find that in health the reflexes are constantly

present in young subjects, that they diminish with advancing years, and may disappear in old age. In nervous disease these reflexes are exaggerated or diminished with the tendon reflexes.

They are unable to decide whether these are bony or aponeurotic reflexes, but they show that they have no relation to the presence or absence of the sense of vibration, and so are not likely to be due to mechanical conduction of vibrations to the posterior roots along bones, as has been suggested. They are similar to the tendon reflexes, and require the integrity of a similar reflex arc.

They point out, further, that percussion over the distribution of a sensory root causes contraction of the muscles innervated by the same segment. D. W. CARMALT JONES.

(33) **THE BABINSKI REFLEX IN EPILEPTIFORM AND APOPLECTIFORM ATTACKS IN THE COURSE OF GENERAL PARALYSIS. (Le réflexe de Babinski dans les ictus épileptiformes et apoplectiformes de la paralysie générale.)** ROBERT and FOURNIAL, *Rev. Neurol.*, November 1906.

THE extensor plantar reflex is extremely rare in general paralysis uncomplicated by any pyramidal lesion: the authors did not find a single definite instance in sixty-six cases. But in seven cases they found a transient extensor response after an epileptic or apoplectic seizure.

They describe these cases in detail; all the patients were typical general paralytics, and in all a flexor plantar response was usually obtained.

Six of these patients had epileptic attacks while under observation, and after each attack an extensor plantar response was obtained for a period varying from two to ten days, after which flexion was again obtained. One patient had a fit, followed by a transient hemiplegia, an extensor response was present on the affected side for one day, after which plantar flexion was again found.

Autopsies were performed in four of the cases, and showed the typical lesions of general paralysis. In several there were definite meningeal adhesions about the motor area.

The authors conclude that the distinction between a transient extensor response, due to cerebral causes, and a permanent one due to organic cause, should be clinically recognised; and further, that pathologically Babinski's sign is not confined to cases of pyramidal degeneration, that pyramidal degeneration of a special kind is the rule in general paralysis, and that Babinski's sign does not occur in general paralysis except as a transient phenomenon.

D. W. CARMALT JONES.

BABINSKI'S PHENOMENON IN MENTAL DISEASE. (Il fenomeno
(34) **di Babinski nelle malattia mentali.)** CASILLO, *Ann. di Nevrolog.*, 1906, Ann. xxiv., F. ii.-iii., p. 181.

1. BABINSKI'S phenomenon is met with in various mental diseases which have an organic basis demonstrated anatomo-pathologically, viz. general paralysis of the insane, epilepsy, and idiocy. In 77 cases of the first of these, an extensor response was obtained in 20 (26 per cent.); in 87 epileptics, 7 times (8 per cent.); in 87 of the last group, 4 times (4·6 per cent.).

2. It is met with exceptionally in some of the autotoxic psychoses, not during the whole course of the disease, but in its acute stage. Its presence may suggest a lesion of pyramidal fibres as a result of the intoxication. S. A. K. WILSON.

THE OCCURRENCE OF BABINSKI'S PHENOMENON AFTER
(35) **INJECTION OF SCOPOLAMINE. (Ueber das Auftreten der Babinskischen Reflexes nach Skopolamin-Injektionen.)** LINK, *Zeitschr. f. klin. Med.*, Bd. 59, H. 2-4.

IN 1903, Volkmann found that an extensor response was obtained during the narcosis produced by injections of morphine and scopolamine, and his observation has frequently been corroborated since. Pawlow noticed an increase in the excitability of cutaneous reflexes in dogs after the injection of scopolamine. The author examined 43 patients, who had already been proved normal subjects from the point of view of reflectivity, while they were under the influence of scopolamine and morphia, and in 37 of these an extensor response was forthcoming. The shortest interval between the first injection and the occurrence of this reflex was 2 minutes; as a general rule, it was obtainable from a quarter of an hour to an hour afterwards, and it often persisted for hours. In 14 cases it was remarked that stroking the sole of the foot produced an extensor response, while the prick of a pin was followed by a flexor one.

Babinski's phenomenon is not by any means an unfailing accompaniment of narcosis in general. On 6 occasions the author was able to note that an extensor response produced by the action of scopolamine disappeared when the patient was subsequently anæsthetised with chloroform. His view is that the presence of this reflex is the sequel to inhibition of cortical action by means of the scopolamine. If it be allowed that a flexor response is a cortical reflex, and an extensor response a spinal one, then it is easy to explain the occurrence of the latter in epilepsy, comatose states, deep sleep, etc.; and further, it can be understood how in

some of the cases of scopolamine injection gentle stroking of the sole was followed by an extensor movement of the great toe, and a stronger stimulus (pin prick) by flexion.

S. A. K. WILSON.

REVISION OF THE QUESTION OF APHASIA, ETC. (Revision de (36) la question de l'aphasie; l'aphasie de 1861 à 1866; essai de critique historique sur la genèse de la doctrine de Broca.) PIERRE MARIE, *Semaine médicale*, Nov. 28, 1906, p. 565.

THE doctrine of the specificity of the third left frontal convolution as the centre for spoken language is based on the first two autopsies performed by Broca. The two brains in question are preserved to this day in Dupuytren's Museum in Paris, where anyone may see them. Now, the first case was undoubtedly a typical motor aphasic, inasmuch as he had hemiplegia on the right side and could say nothing whatever beyond the syllable "tan." The pathological condition is a gross softening which has involved the third left frontal convolution in its posterior half, the Rolandic convolutions in their lower ends, the greater part of the upper temporal convolution, and a very considerable portion of the supra-marginal gyrus!

In the second case the patient was 84 years of age and had difficulty in speaking. An examination of his brain shows absolutely no focal lesion of the third frontal convolution, but merely a senile atrophy of some of the gyri in that area, with depressions at intervals where the atrophy is more pronounced. This condition is far from uncommon in senile cerebra, as Léri has recently emphasised. In Marie's opinion, therefore, Broca's second case is not one of aphasia at all, but almost certainly a "lacunaire," *i.e.* a case in which numerous lacunes of cerebral disintegration existed in the grey matter of the great ganglia.

Marie again directs attention to the fact that the artery which supplies the third frontal convolution is a small recurrent branch from the middle cerebral, and therefore unusually prone to suffer if the latter is involved at all, which explains the frequency with which softening of that convolution is associated with softening of other perisylvian gyri. From the teleological standpoint, it is unlikely that a convolution with so precarious a blood supply should have assumed so important a rôle as that of being the centre for spoken language. The rest of the article consists of a most interesting historical retrospect on the teaching in neurology of the years succeeding 1861. It is clearly shown how deeply current thought was influenced by the tenets of Gall and other phrenologists, that the frontal lobes were the seat of the faculty

of language, and how Broca was unable entirely to rid himself of the conception. The original must be consulted to follow the steps whereby the doctrine of the localisation of the motor speech centre in the third left frontal convolution—a doctrine never fully substantiated or maintained by Broca himself—has become an indispensable part of neurological teaching, although in Marie's opinion it is based on absolutely erroneous conceptions.

This opportunity may be taken to refer to the admirable lucidity and charming style of Professor Marie's articles on aphasia. It may be said of them, as he himself says of Broca's work, that they are "un modèle de clarté, de bonne humeur, d'esprit, et de courtoisie." S. A. K. WILSON.

CASE OF CONGENITAL WORD-BLINDNESS (INABILITY TO LEARN TO READ). (37) J. HERBERT FISHER, *Ophthal. Rev.*, Nov. 1905.

VERY few cases of this condition have been recorded, but probably many have been overlooked and treated for refractive errors. The patient was a girl aged six years, of normal intelligence and with no defect of vision for ordinary purposes. She had been trying to learn to read for two years but had made little progress. The letters became mixed up and she mistook simple words. She had also great difficulty in adding figures and in reading music. The fact that her mother's brother was ten years of age before learning to read, and in adult life was said to spell abominably, indicates a family tendency to imperfection in development of the visual memory centre for words in the cortex of the left angular gyrus. The patient made good progress under the "look and read" system —learning to recognise words as a whole and not by their individual letters. The writer considers this the best method of instruction for such cases. Spelling should be taught orally, and the use of a typewriter might help to save blunders in spelling later in life. HENRY J. DUNBAR.

A CASE OF TACTILE APHASIA. (A propos d'un cas d'aphasie tactile.) (38) M. NOICA, *Rev. Neurolog.*, Nov. 30, 1906.

THE first part of the paper consists of a review of the views held as to tactile aphasia by Raymond and Egger on the one hand, and Déjerine on the other. The former, at a meeting of the Société Neurologique, had showed a case of a lesion in the left hemisphere, as a result of which the right arm alone was involved. This patient, with his eyes closed, could tell, by means of the right hand, various attributes of different test objects, but was unable

to give them a name. Raymond and Egger concluded that, as the power of tactile sensibility was not lost, the syndrome they describe does not depend on loss of such sensibility, and is therefore a true "tactile aphasia" depending on a central lesion, and is comparable to word-deafness.

At the next meeting of the Society, Déjerine showed a man similarly affected, who exhibited exactly the same phenomena. Déjerine was, however, of the opinion that there was a diminution in the acuity of tactile sensation in his case, and stated in his opinion that no man, intellectually sound, can present signs of "tactile aphasia" unless he has some disturbance of tactile sensation. Claparède, though not supporting the name, supports the theory which Raymond and Egger hold, and believes the essential lesion to be a central one, affecting the association fibres between the centres of tactile perception and speech.

The latter part of the paper contains an account of a case of traumatic ulnar nerve neuritis under Noica's personal observation. This patient exhibited the same "tactile aphasia" as already described, but only in the third and fourth fingers, supplied, of course, by the injured nerve. He was readily able to name any test object placed in contact with the remaining portion of his hand.

The fault in this case was obviously not central but peripheral, and supports Déjerine's views on the subject.

C. M. HINDS HOWELL.

PSYCHIATRY.

OCCUPATION PSYCHOSES. (**Berufspsychosen.**) W. HELLPACH (39) (of Karlsruhe), *Psych.-Neur. Wchnschr.*, July 21, 28, Aug. 4, 1906.

GENERAL considerations on the peculiar mentality developed by or shown in certain occupations. The author thinks that the collector, with his hobby warping his mental activity, has a close affinity with the litigiously insane (Querulanten). Hysteria dominates the occupation psychosis, or psychopathy, of the actor; this profession involves the abrupt changes of mental attitude and of the group of dominant interests, and an unusual command of mimic innervation, and, as a consequence, an abnormal facility of discharge along these paths—an important factor in the genesis of hysterical complexes. Hypochondria is the special psychopathy of the medical profession, which involves, like the actor's, the living of oneself into the life of another individual, and in addition, the introspective analysis of one's own abnormal instincts as a guide to those of others, and the ability to see in the earliest symptoms the first signs of developing disease.

Mental disorders depend on the two factors, constitution and environment, and the nature of the latter, due to a special occupation, may be the upsetting factor; with our knowledge of the constitution of a person, certain occupations may be looked on as involving certain dangers from the psychopathic point of view.

C. MACFIE CAMPBELL.

THE MOTOR DEGENERATIVE SEQUENCE IN DEMENTIA.

(40) H. A. TOMLINSON (of St Peter, Minnesota), *Journ. Am. Med. Assoc.*, Oct. 27, 1906.

THE author believes that the variation of muscular activity following the loss of mental capacity has been considered too little. Motor impulses are centrifugal and have their basis in the liberation in consciousness of impressions that have pre-existed and given rise to them. There are few forms of motion which come as a direct response to external stimuli. In the processes of degeneration the forms of motor activity involved in the most abstract forms of expression are the first to be lost, and the failure shows itself in the co-ordinated movements which are last in the order of development. When mental reduction proceeds until memory and attention are involved, there is commonly some evidence of relaxation in the normal tension of the facial muscles and the muscles of the eyeball; next the finer movements of hand and arm are affected, and this is commonly as far as the degenerative changes go unless mental reduction becomes extreme. These degenerative changes are somewhat similar in senility and in dementia, but the paresis of dementia is commonly spastic, while in senility it takes the form of tremor. Finally, the author believes that, where the motor activities are involved in the order of their development and follow this order, the change will be a permanent one. When this order is departed from and is not symmetrical and progressive, there is a probability that the power will return more or less completely.

C. H. HOLMES.

ON SO-CALLED MORAL INSANITY. (Einige Punkte aus der Lehre der sogenannten "Moral Insanity.")

(41) P. NÄCKE (of Hubertusburg), *Psych.-Neur. Wchnschr.*, June 23, 30, 1906.

THE author takes exception to the views of the Italian school, which makes in the group of habitual criminals a large sub-group of born criminals; he holds that the born criminal is a rarity, and that the influence of the environment has been underrated by Lombroso and his followers. The habitual criminal is the product of a particular constitution and exogenous influences, and

the variations in the latter form the important element in the majority of cases. The essence of the criminal constitution lies in a defect of that affective element which gives to ethical conceptions their dynamic value; this affective defect is closely connected with fundamental instincts and impulses, and in this connection the author makes some suggestions of anatomical correlation. C. MACFIE CAMPBELL.

PSYCHOPATHIC TRAITS IN ALIENISTS. (Psychopathische Züge
(42) **bei Irrenärzten.)** G. LOMER, *Psych.-Neur. Wchnschr.*, Aug. 11, 1906.

A RATHER pessimistic view of the effect of the work and environment on the mind of the hospital physician. Psychiatric work requires keen sympathy and an introspective turn of mind, which qualities are found only in the highly-organised individual, who is more susceptible than the average man to the influence of constant association with what is æsthetically and ethically abnormal. The tendency to carry the analytical and introspective atmosphere of the ward into one's whole life results in a morbid attitude. C. MACFIE CAMPBELL.

TRANSITORY PSYCHOSES DUE TO ALCOHOLIC INTOXICA-
(43) **TION. (Les ivresses délirantes transitoires d'origine alcoolique.)** E. DUPRÉ (of Paris) and R. CHARPENTIER (of Paris), *L'Encéphale*, Jan. 25, 1906.

TWO cases of transitory psychosis on the basis of acute alcoholic intoxication. The first case was a temperate boy of 19, worn out by overwork and worry, who unintentionally drank a small quantity of rum which had been put in a glass of beer; six hours later he entered the police station with a large butcher's knife and said that he had just killed six Prussians in his room and that there remained twenty to be killed; he spent the night in the hospital, slept soundly, was astonished next morning to find himself in the hospital, had absolutely no memory of what he had said on the previous evening. He was perfectly clear and was discharged.

The second case was complicated by chronic alcoholism, and there was a history of a previous hallucinatory attack. Patient was a man of 40, who, after two days excessive drinking, entered the police station and accused himself of imaginary crimes. Next morning he was clear, recognised that he had talked foolishly the previous evening under the influence of drink, was able to be discharged. Such attacks in their onset, duration, and termina-

tion are quite different from attacks of sub-acute alcoholic delirium, on the basis of chronic alcoholism.

C. MACFIE CAMPBELL.

THE BLOOD PRESSURE IN PARESIS. G. L. WALTON (of Boston), (44) *Journ. Am. Med. Assoc.*, Oct. 27, 1906.

FROM the reports of observations by Pilcz, Alexander, Craig, and others, the hope has been raised that in blood pressure there lies a practical aid in the diagnosis of paresis from other nervous diseases. Many more observations must be carefully made and tabulated before such a conclusion can be drawn. Walton examined 108 male patients suffering from this disease; cardiac, renal, and arterial disease was absent in but 44 of them. He arrived at the following conclusions: (1) The average blood pressure in paresis, taken as a whole, is high; (2) this is doubtless due to the prevalence of atheroma, with its cardiac and renal complications; (3) the average blood pressure in cases of paresis without atheroma, cardiac enlargement, or renal disorder, is probably somewhat lower than that in health, but the variations are so great that it cannot be said to be uniformly low; (4) the test is not likely to prove of great value in the differentiation of paresis from other nervous disorders, though here, as elsewhere, useful in estimating the circulatory condition of the individual; (5) these observations are too few to establish a rule with regard to the blood pressure in varying emotional states. As far as they go, however, they tend to show (*a*) that the excited states of paresis are as likely to be accompanied by high as by low pressure; (*b*) that mental depression is accompanied by high oftener than by low pressure, but that it is not incompatible with low pressure; (*c*) that while the average pressure in euphoria is perhaps somewhat lower than in other mental states of the general paralytic, it is not inconsistent with high pressure, or with pronounced atheroma with cardiac and renal accompaniments. C. H. HOLMES.

ON THE APPLICATION OF ARTIFICIALLY PRODUCED VENOUS
(45) **HYPERÆMIA OF THE BRAIN IN MENTAL DISORDERS. (Ueber die Anwendung künstlich erzeugter venöser Hyperämie des Gehirns bei Geisteskrankheiten.)** NEU (of Galkhausen), *Psych.-Neur. Wchnschr.*, July 7, 1906.

BIER found that by artificially causing venous congestion of the brain he caused improvement in some cases of epilepsy, chorea minor, and headache of various origin. Neu, following Bier, has

treated cases of mental disorders by the same method. To produce the congestion he applied an elastic bandage to the neck, increasing the pressure gradually to avoid sudden variations of blood pressure, and lowering the pressure of the bandage on complaint of pressure or heaviness in the head. The period of congestion was usually under twelve hours. Twenty-three cases were thus treated: general paralysis, 3; epilepsy, 3; melancholia (not further defined), 8; dementia præcox, 3; paranoia, 5; amentia, 1. In no case was the treatment injurious to the patient; the case of amentia was cured. In the cases of epilepsy, dementia præcox, and paranoia no improvement was observed. Some improvement was obtained in all the cases of melancholia. By the same treatment very marked relief was given to three cases of anæmic headache. The author does not claim more, from the small number of cases treated, than to show the desirability of trying this method of treatment in cases of mental disorder. C. MACFIE CAMPBELL.

SCHEFFEL: HIS DISEASE. (**Scheffels Krankheit.**) Dr P. J. (46) MÖBIUS. Pp. 40. Halle a. S.: Carl Marhold. 1906. Price 1 Mk.

THE first half of this pamphlet contains an account of the medical history of Joseph Victor Scheffel, the poet, who was born at Karlsruhe in 1826, and died at the age of sixty of cardio-vascular disease. Hereditarily burdened, dogged by ill-health from the age of twenty-seven, when he suffered his first cerebral attack, the subject of repeated relapses, culminating in his thirty-fifth year in one marked by delusions of persecution, a morbid sense of culpability and extravagant conduct; driven from place to place in search of health, a compulsory vagabond, his undoubted genius clouded by fears of a relapse, or obfuscated by alcoholic excess, his output of work is as remarkable as his eventual, if tardy, recovery, qualified though it was by a partial dementia which removed the poet, if it left the man. In reviewing the whole case, the author comes to the conclusion that Scheffel was the subject of dementia præcox; but however this may be, his brief and necessarily fragmentary description of a deeply interesting personality will be of value to students of the pathography of genius. Dr Möbius' brochure is in continuation of his previously published account of Robert Schumann, and the second half of the present pamphlet contains a critical appreciation of the former work, questioning Dr Möbius' diagnosis, by Dr Gruhle, of Heidelberg, and Dr Möbius' reply thereto.

R. CUNYNGHAM BROWN.

TREATMENT.

THE "MOUNTAIN-CURE" OF NERVOUS DISORDERS. (Über die Indicationen der Hochgebirgskuren für Nervenkranke.)
(47) A. NOLDA (St Moritz), *Abhand. a. d. Geb. der Nerven- und Geisteskrankheiten*, Bd. vii., H. 3, p. 16. Halle a. S.: Carl Marhold. 1906.

AFTER many years spent in extensive practice in Alpine resorts at high altitudes, Prof. Nolda is able to speak with authority on the results of treatment of the neuroses at those levels. His material comprises over 3000 nervous cases, over 2000 of whom were neurasthenics, but also many cases of hysteria, mild melancholia, Graves' disease, and a small number of epileptics. The best results, he finds, are obtained in recently acquired neurasthenia, the symptoms of head pain or pressure, sleeplessness, lassitude, and cardiac arythmia disappearing within, at most, fourteen days, headache and vertigo often on the first day. About 90 per cent. of all cases of neurasthenia are improved by mountain treatment.

Heart disease, with angina pectoris, or severe myocardial degeneration, uncompensated valvular disease, pronounced dilatation, or hypertrophy, are, of course, important contra-indications; but he maintains, in opposition to Krafft-Ebing and Löwenfeld, that mild cases of "cardiac or vaso-motor neurasthenia" are much improved by removal to high altitudes. Another affection giving remarkably good results is that of hay-fever, about 70 per cent. finding immediate relief. An interesting point brought out by the author is that all medical treatment requires the most careful application at these heights, drugs of all kinds, particularly opium and its alkaloids, having a much more pronounced effect than at lower levels, and requiring exhibition in very small doses.

R. CUNYNGHAM BROWN.

Reviews

STUDIES IN CLINICAL PSYCHIATRY. LEWIS C. BRUCE, M.D., F.R.C.P.E. Pp. 246. London: Macmillan and Co., Ltd. 1906. Price 10s. 6d. net.

THE studies in clinical psychiatry which form the basis of this work are specially directed towards the physical disorders which so frequently accompany mental disease. Such physical disorders embrace changes in the processes of digestion, of assimilation, and of excretion—changes in the clinical composition of the blood, and in the number and relative proportion of the formed elements, changes in the cardio-vascular system and sometimes changes in various glands. In some groups of mental disorder the nature of the physical changes suggests the possibility of disordered metabolism being at the bottom of the mischief; but while hypotheses on this subject have been formed with great prodigality, the results of the workers in this field are extremely fragmentary and often conflicting, and do not warrant any definite conclusion as to the exact rôle played by these factors in the mental disorder. Any contribution to the elucidation of this question is therefore very welcome. Dr Bruce has in his studies paid particular attention to the condition of the blood and to the excretion of urea, and also to variations in the blood pressure. He has also made frequent bacteriological examinations of the blood, and has studied the agglutinines found in some conditions. His results are interesting, and must prove a stimulus towards further bacteriological work in psychiatry.

In 10 cases of catatonia the blood was examined bacteriologically "during the acute stage"; one case presented a pure culture of a short streptococcus; the serum reaction of 20 cases of catatonia to this organism was tested, and agglutination was obtained in 15 cases, while 20 control serums did not give agglutination. Of 120 cases of other forms of insanity, 12 gave agglutination; these 12 cases were rather heterogeneous, but apparently all were conditions of excitement—3 cases of circular insanity, 2 of excited melancholia, 2 of alcoholic mania, 2 of mania with confusion in adolescent cases, 1 hebephrenia, 1 senile, 1 unclassified. Unfortunately the author does not record the clinical history of the case from which the organism was isolated, and does not say whether there was any intercurrent malady or not. He also omits any description of the cases which gave agglutination to this organism. In the absence of such absolutely necessary clinical data, this

isolated observation cannot be utilised for any discussion of the nature of catatonia and its general ætiology. In the chapter on catatonia, the author states that in the first stage of the disease there is a moderate persistent hyperleucocytosis; at the termination of the acute stage, and just prior to the onset of stupor, there is a sharp rise in the leucocytes; on the onset of stupor, the leucocytes may fall, but immediately rise again, averaging between 12,000 and 16,000 per c.mm. One cannot appreciate correctly the value of such observations unless there is given an opportunity to study the clinical histories upon which the results are based, or unless there is some guarantee that the author is referring to some well-defined clinical group. The fact that under "acute melancholia" he gives a series of heterogeneous cases, belonging in reality to widely different groups, raises the question whether under catatonia he has not also grouped together widely dissimilar cases. It is certain that in the catatonic phase of a deteriorating psychosis, leucocytosis does not always occur. In some cases with leucocytosis a broncho-pneumonic attack or some other intercurrent disease explains the leucocytosis. In any case, in considering the leucocytosis when present, one must keep in view at the same time the possibility of intercurrent disease. The fact that so many of the catatonics examined yielded an agglutinin to this organism is certainly one fact contributed towards the demonstration of the possibility that a catatonic symptom-complex may be associated with a definite toxæmia. Dr Bruce concludes that catatonia is an acute toxic disease with a definite course and onset. Such a conception is difficult to reconcile with what we know of the whole development of cases of a deteriorating psychosis which presents a catatonic phase. One may note that in the three cases which are given as examples of the psychosis the leucocytosis on admission was not above 11,000, and that in two cases the marked hyperleucocytosis came on during the period of stupor when there is a special tendency towards the development of broncho-pneumonic attacks, which, unless they are specially looked for, may easily escape attention.

Another interesting observation from the bacteriological point of view is that of a woman, aged 40, who died during a second attack of delirium. Patient had a leucocytosis of 30,000; a culture from the bone marrow and from the lungs gave a pure growth of a coccus, while the other organs were sterile.

It is also interesting to note that Dr Bruce found that 11 out of 14 cases of circular insanity agglutinated an organism isolated from the blood of a case of excitement with confusion.

To draw conclusions from such observations would be premature, and the observations with regard to leucocytosis are frequently at variance with those of other workers. One must accept the

conclusions with the greatest reserve in view of the extremely unsatisfactory observations of the author on his first group of insanities of toxic origin, viz. "insanities the result of toxines of metabolic origin." Under this group he includes (*a*) acute melancholia, (*b*) thyroidogenous insanity, (*c*) a variety of puerperal insanity, (*d*) delusional insanity, (*e*) chronic metabolic toxæmia (premature senility, chronic brain atrophy). "Acute melancholia is a definite physical disease with definite physical symptoms, and it is a disease of adult life and the decline of life . . . the diagnosis depends upon the clinical symptoms—the disordered alimentary tract, the rapid pulse, the high arterial tension, the deficient excretion of urine and urea, and the absence of hyper-leucocytosis." Such symptoms do not look promising for purposes of clinical differentiation, and one is not surprised, under such circumstances, that the group of acute melancholia contains the records of cases belonging to absolutely different groups. Four charts are given to illustrate the temperature, pulse, arterial tension, and excretion of urea which are claimed as typical of acute melancholia, but in these charts the clinical symptoms upon which the diagnosis is said to rest are not prominent: one does not note a rapid pulse; there is no characteristic temperature; the high tension during a period of agitation is what one might expect; and the diminished excretion of urea is left alone to support the weight of the diagnosis. The first case is a typical example of the depressed phase of manic-depressive insanity, the essence of which is a simple sadness—retardation complex—and the prognosis of which is good for the individual attack, but with liability to the recurrence of a similar depression, or of an "equivalent" manic attack (not merely a maniacal excitement, but an attack of a form as well characterised as the depression). Of the next two cases practically nothing but the charts is given. The fourth case presents a condition of hallucinatory confusion, such as frequently follows conditions of exhaustion, and which is different in onset, course, and prognosis from a case like the first. This case of hallucinatory confusion may with advantage be compared with a case recorded (p. 52) as an example of insanity of exhaustion, grouped under "insanities of non-toxic origin." The case is described as depressed and confused; arterial tension 140 mm., T. 99, skin, as a rule, dry, excretion of urea not mentioned. Why is this case looked upon as an insanity of non-toxic origin when, in regard to the manic-depressive case above recorded, "the dry pigmented skin, the deficient excretion of urine and urea, the slightly febrile temperature, unaccompanied by a hyperleucocytosis, combined with depression and slight confusion, are typical of a mild attack of metabolic toxæmia?" It would be superfluous to cite other examples to show the absurdity of classifying mental cases

according to the present fragmentary knowledge of their disordered metabolism. Studies in metabolism are of great importance, but they must be more than mere fragments before they can be used for the clinical grasp of cases; and the complex conditions must be always kept in mind, not to mention the more elementary facts, such as the relation of excretion to food taken in.

Psychiatry has made a great advance in leaving the mere symptomatological analysis of mental states for the better standpoint from which the onset, course, symptomatology, and outcome are all utilised for the appreciation of the nature of the disease; order has thus been introduced into a most intricate subject by this method, the "clinical method" *par excellence*, which consists in the unprejudiced observation of each pathological phenomenon whatever its nature, whether it be flight of ideas or deficient excretion of urea, and in the analysis not only of a transitory clinical picture, but of a continuous pathological process. The author says in the preface, "when psychology is divorced from psychiatry, and the study of psychiatry is prosecuted along the lines of advance in general medicine, our knowledge of mental diseases cannot fail to be added to." Medicine, however, has not made progress in any branch by neglecting to study the disordered functions of the special system which is its subject matter, and psychological phenomena are as much part of human biology as the phenomena of nutrition.

It is through refusing to adopt the "clinical method" that so little progress has been made of late by British psychiatry. If the author of these studies had followed this method, or taken advantage of the advances made by those who have followed it, the studies would have remained fragmentary, but the fragments would have been more easy to fit in with those provided by other workers for the building up of a pathology of mental diseases.

C. MACFIE CAMPBELL.

ANALYSE VON 200 SELBSTMORDFÄLLEN. By Dr HELENE-FRIEDERIKE STELZNER. Pp. 124. Berlin: S. Karger. 1906. Price 4 marks.

THE 200 cases dealt with in the book are all women who were admitted into the female division of the Psychiatric Department of the Charité Hospital, Berlin, between the years 1892 and 1905. While the greater proportion of these cases belonged to the working classes, they included representatives of all social classes, as well as of various religious denominations and nationalities. A further important point in this connection is that, in addition to the fact that the larger number were labouring under some form of mental

disease, there were 31 patients in whom no antecedent or subsequent mental aberration could be discovered. The following classification shows the mental condition of the patients on examination. Melancholia, 65 cases; paranoia (acute and chronic), 35; psychopathic constitution (degenerate), 32; without any apparent mental symptoms, 31; epilepsy, 11; dementia præcox, 7; general paralysis, 6; alcoholism, 6; senile dementia, 4; imbecility, 3; circular insanity, 1.

The author does not profess to add anything new to the already extensive statistics of suicide from a demographic point of view, but merely to present an analysis of the cases observed, with the object of inquiring into—(1) the manner in which mental affections conduce towards suicide; (2) the course and prognosis of mental affections complicated by suicidal attempts; (3) observations on suicide occurring in persons of apparently sound mental health; (4) the mental antecedents of persons who attempt suicide; (5) a discussion of the different methods of committing suicide; and (6) a consideration of the question of heredity as it affects suicide.

Judged entirely from the stated objects of the book, it must be admitted that Dr Stelzner has succeeded in presenting, out of very ordinary material, some interesting and important results. The value of these results has been enhanced by the pains which have been taken to enquire into the antecedents, physical, mental, and social, of each patient; into the motives which actuated the attempt, as well as into the after-history of the cases. In the great majority of the cases the attempts at suicide were initiated and suggested by mental depression either pathological or induced by adverse circumstances in the surroundings. Of the 169 cases of mental disease, 65, or 38 per cent., were suffering from melancholia, but these do not exhaust the mental depressive states in the other forms of insanity mentioned which directly influenced the act. Thus, for example, all the six patients labouring under general paralysis attempted suicide in the depressed stage of the malady which is now so frequently observed. The suicidal attempt was the first observed symptom in 13 out of the 65 cases of melancholia, but the author correctly observes that that fact does not necessarily prove that the disease did not exist prior to the attempt. For, as he points out, the patients largely came from a class wherein the struggle for daily bread or the exigencies of social circumstances left little time for the observation of the slighter mental or physical ailments. After the shock of the attempt and the relaxation from other engrossing cares had broken down self-control and given time to think subjectively, the patients developed the usual agonising symptoms of the disease. A very interesting point is that out of 18 cases of climacteric melancholia, practically

all recovered. This is a better prognosis than is usually given of this form of the disease, even in the absence of distinct attempts at suicide. For the treatment of degenerates who have attempted suicide, as well, we presume, as for all degenerates who are a nuisance to society, the author strongly urges the establishment of special forms of institution, where not only care but training and education of a moral nature should be given. A feature of the statistics which deserves notice is the small number of suicides due to alcohol. This is directly contrary to the opinion of some who hold alcohol to be one of the most active agents in developing the impulsive and mental attitude which leads to self-destruction. Of the 31 cases of suicide without any antecedent or subsequent symptoms of mental disease, it is interesting to note that heredity to suicide is not mentioned in one case. Four patients showed heredity to the psychoses, and 10 were the subjects of one or more of the milder neuroses. Suicide is generally regarded as one of the most hereditary conditions, and it is not a little surprising to find that even out of such a small number as 30 cases no direct heredity exists.

Dr Stelzner has broken new ground by means of her treatment of the subject and by reason of the many suggestive facts she has been able to elucidate from an analysis of her cases. The book is one which everyone interested in suicide, whether from the clinical or the social points of view, ought to read.

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Review
of
Neurology and Psychiatry

Original Articles

CIRCUMSCRIBED HÆMORRHAGIC CORTICAL ENCEPHALITIS, WITH THE REPORT OF A CASE IN WHICH THE LESION WAS LIMITED TO THE MOTOR ZONE, THE CHIEF CLINICAL MANIFESTATION BEING JACKSONIAN EPILEPSY.[1]

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CIRCUMSCRIBED cortical encephalitis studied clinically, and its existence demonstrated by necropsy and microscopical examination, while not unique, is still so rare as to give value to the report of a new case. I shall not go into the history and literature of the subject, which is made unnecessary by the valuable articles of Oppenheim,[2] Hoppe,[3] and others. The entire

[1] Read before the meeting of the American Neurological Association, held in Boston, June 4 and 5, 1906.

[2] Oppenheim, H., on "Die Encephalitis und der Hirnabscess," vol. ix. of Nothnagel, "Specielle Pathologie und Therapie," Wien, 1897. Oppenheim has a good practical article in his text-book on "Diseases of the Nervous System" on the different forms of acute non-purulent encephalitis.

[3] Hoppe, H. H., "Acute Hæmorrhagic Cortical Encephalitis," *Journ. of Nerv. and Ment. Disease*, vol. xxix., No. 9, September 1902.

subject of non-purulent encephalitis, cortical and basilar, has received most attention from German writers, although a few excellent articles have been written by French, English, and American neurologists, that of Hoppe being the most elaborate contribution, so far as I know, of anyone writing in this country.

The impulse given to the study of cortical encephalitis by Strümpell[1] is a good illustration of the value of the application of deductive reasoning in the development of exact knowledge. Strümpell, largely on theoretical grounds and on the basis of analogies, advocated the view that certain of the infantile palsies of children were due to a cerebral polio-encephalitis comparable to the polio-myelitis which had long been the well-known most important cause of the spinal infantile paralysis of children. The truth of his deduction has since been absolutely demonstrated, although as yet only in a limited number of cases.

For the part played by Leichtenstern, Koenigsdorf, Schmidt, Raymond, and others in the history of the subject, the reader is referred to the monograph and to the articles of Oppenheim and Hoppe above cited.

The first case of circumscribed hæmorrhagic encephalitis, cortical and subcortical, with necropsy and microscopical examination, recorded in this country was by Dana and Schlapp[2] before the meeting of the New York Neurological Society held January 2, 1900. The patient was a man sixty-seven years old, in whose blood the malarial plasmodium was found. After headache he had a hemiplegic seizure. His temperature was at first 104° F., but had subsequently fallen, rising again before death. His chief focal symptoms were left hemiplegia almost complete, immobility of tongue, and marked oral paresis. After he had been in the hospital for a week the paralysis of the arm and leg disappeared, but that of the tongue and lips persisted. No sensory symptoms were noted. An area of hæmorrhagic softening was found in the lower portion of the post-central gyre and in the marginal gyres, involving mainly the deep part of the upper lip of the Sylvian fissure. Two or three smaller areas of hæmorrhage were found in the centrum ovale. These were the

[1] Strümpell, A., *Magdeburger Naturforscher Versammlung*, Sept. 22, 1884.

[2] Dana, C. L., and Schlapp, M. G., *Journ. of Nerv. and Ment. Disease*, vol. xxvii., No. 2, February 1900.

only brain lesions, but the spinal cord showed two polio-myelitic foci, one in the upper and the other in the lower portion of the thoracic cord. Microscopical examination of the area of cerebral disease showed dilatation of blood-vessels, extravasations of blood, and great proliferation of cells. Small areas of softening were also present in the neighbourhood of the hæmorrhagic extravasations. The appearances closely resembled those described as encephalitis hæmorrhagica. The case had some interesting features in focal symptomatology. The paralysis of the tongue and lips and the site of the lesion pointed to the existence of centres for these parts in the grey matter of the roof of the Sylvian fossa. Dana and Schlapp suggest that the case might be classed under the head of degenerative hæmorrhagic encephalitis. Their examination of the autopsy records of sixty-seven cases of apoplexy showed that forty-two of them were hæmorrhagic, eleven embolic, eleven thrombic, and three encephalitic. The case recorded by them is of interest in connection with that which will be presented in this paper, because of its focal motor symptomatology and the circumscription of the lesion. Jacksonian epilepsy was not present, but cortical or cortico-subcortical paresis and paralysis were marked symptoms.

Harlow Brooks[1] reviews the subject of acute non-suppurative encephalitis in an interesting manner. He believes that a considerable number of cases which recover are regarded as hysteria. The lesions of the disease are enumerated by him as: (1) Cerebro-spinal meningitis, probably secondary to cerebritis and of a "cellular type." (2) General non-septic cerebritis affecting all parts of the cerebrum, and most marked in the cortex and particularly so in that of the motor areas. (3) Degeneration of many of the ganglion cells of the cortex. (4) Degeneration of many of the fibres arising from the large pyramidal cells of the cortex. (5) Diffuse degeneration affecting many of the fibres passing through both internal capsules. (6) Similar changes in the cerebellum. (7) Degeneration of many of the tracts of the pons and oblongata. (8) Degeneration of the chief descending tracts of the spinal cord. (9) Slight changes of the cytoplasm of the ganglion cells of the anterior horns of the spinal cord, probably secondary. The symptoms which caused

[1] Brooks, Harlow, *Medical News*, Aug. 8, 1903, vol. lxxxiii., No. 6.

these conditions in the case reported by Brooks were coma, stertorous respiration, absent patellar reflexes. The unconsciousness persisted for three weeks. Necropsy showed no gross organic lesions.

Cases of acute circumscribed hæmorrhagic cortical encephalitis of the motor zone with Jacksonian epilepsy as a part of their symptomatology are very rare, and yet one of the first cases of this affection recorded had among its symptoms both cortical epilepsy and hemianopsia. This case was recorded by Fürbringer.[1]

Batten,[2] who has discussed the subject of cortical encephalitis in a most interesting manner, especially dwelling upon its relations to polio-myelitis and to encephalitis involving the basal nuclei, has recorded a case with necropsy in which a boy nine years old had aphasia of motor type and right hemiplegia, the eyes and head being turned to the left. Consciousness was at first not lost, but the child soon became drowsy, cried out occasionally, and put his left arm to his head as if in pain. Jacksonian spasm was not present. The coma deepened and the patient died three days after the onset of the disease. Microscopical examination of the motor region showed numerous small hæmorrhages and thromboses of the smaller vessels; but although this condition was most marked in the motor region of the cortex on the left side, it was not limited to that region. In this case, as in that of Fürbringer, the affection was attributed to influenza.

In another case without necropsy, recorded by Batten, a boy two and a half years old, the patient had for a time slow rhythmical movements of the left arm and leg. These later passed off and similar movements occurred in the right arm and leg, the latter persisting but becoming gradually less and less marked. Other focal symptoms were paralysis of the left external rectus, ataxia preventing him from walking, and intention tremor in the right arm and leg. The right knee-jerk was more active than the left; the right plantar reflex gave flexion and the left an extensor or Babinski response. This patient was taken ill in August with fever and unconsciousness which lasted for two days; he eventually recovered.

It is difficult, as Batten says, to locate the lesions causing the

[1] Fürbringer, P., *Deutsch. med. Woch.*, No. 3, Jan. 21, 1892.

[2] Batten, Fred. E., *The Lancet*, Dec. 20, 1902; and *Trans. of the Med. Soc. of Lond.*, vol. xxviii., p. 116, 1905.

symptoms. The encephalitis probably affected both the cerebrum and cerebellum as well as the sixth nerve in its course after leaving the nucleus. Whether the rhythmical spasm in this case was due to cortical motor lesions cannot be asserted. It sometimes happens, as the writer has shown in another paper on Jacksonian epilepsy,[1] that local spasm may be due to lesions at the base, as in two cases of cerebello-pontile tumour recorded by him. The probable explanation of such cases is that they are caused by irritation of the pyramidal tracts when the arm and leg are involved, or of the facial tract or the nerve itself when the face takes part in the spasm. In the case of Batten, as both cerebellar and cranial nerve symptoms were present, the rhythmical movements may have been due to involvement of the motor tracts in the pons and oblongata; on the other hand, as the lesions of polio-encephalitis are often found in several or many parts of the brain, the motor cortex or subcortex on both sides may have been the seat of circumscribed foci of encephalitis.

Spielmeyer[2] has contributed an article on the "Symptomatology of Cerebral Encephalitis, especially on an Epileptic Form of this Affection." This article, or a portion of it, is of considerable interest in connection with the case here recorded. According to him, either coma, paralysis, or motor excitation is always present in this affection. His group of epileptic cases, which is of particular interest here, is somewhat fully discussed. The spasms may be either Jacksonian or general, and are usually transient, but may disappear to return. I have seen several illustrations of this form of focal encephalitis in children in which the spasms, having ceased, returned, or in which they did not appear until some years after the attack of encephalitis, this having presented chiefly the features of the comatose or paralytic variety. Two personal cases are given by Spielmeyer showing marked encephalic lesion both of the cortex and subcortex of the motor region.

Of interest is the view taken by Spielmeyer that the disease which the epileptic form of encephalitis most counterfeits is brain tumour. The mode of onset and progress of the two affections

[1] Mills, C. K., *The Boston Medical and Surgical Journal*, vol. cliv., No. 17, April 26, 1906.

[2] Spielmeyer, W., *Centralblatt für Nervenheilkunde und Psychiatrie*, vol. xxvii., June 1904.

and the presence of optic neuritis in brain tumour furnish the best points of differentiation. The frequent occurrence of focal encephalitis in connection with infectious diseases is also a point of much diagnostic value. Attention is called to the resemblance of the symptoms of epileptic encephalitis to those of "meningitis serosa," and it is suggested that as cases of the latter affection have not been verified by necropsy, this form of so-called meningitis may be in reality an encephalitis.

Jacksonian Spasm involving the face and arm—facio-brachial monoparesis—no impairment of sensation—focus of hæmorrhagic encephalitis in the motor zone, sharply delimited posteriorly by the central fissure.

M. G., eighty-three years old, was a patient in the writer's wards of the Philadelphia General Hospital. She had been for nearly three years before her admission, October 12, 1905, an inmate of the out-wards. Her family and past history were negative, except the record of some heart disease which had existed for about a year. The illness for which she was brought to the hospital began a few days before her admission. She had three attacks of spasm involving the left side of the face and left arm. She was not unconscious during the convulsive seizure, but the left arm became weak, especially at the wrist.

Examination showed an old woman well nourished. Her tongue protruded towards the left side. The left side of the lower face was smoother than the right, and voluntary movements were not as well performed on the left as on the right. The movements of the upper half of the face were well performed on both sides, but the ocular movements were not well executed, either singly or together.

The left ocular and palpebral conjunctivæ were greatly inflamed. The pupils were moderately dilated, round, and responded well to light, accommodation, and convergence. The left upper extremity showed distinct weakness in all movements with wrist-drop. The patient could not elevate the hand nor extend the fingers. Flexion of the fingers and hands was likewise impossible, but the biceps and triceps movements and those of the muscles of the shoulder girdle were preserved to a slight degree. No paralysis was present in the lower extremity.

Sensation was normal in the paralysed parts and also elsewhere throughout the body. The reflexes were normal.

During the time of examination the left side of the face and the left hand were the seat of intermittent convulsive movements. The hand was drawn somewhat to the left and the angle of the mouth was drawn outwards and upwards. The eye was closed and the brow wrinkled, but without any frowning movement or movements of the eyeballs or jaws. Dilatation of the left nostril and movement of the platysma took place. In the spasm the movement of the hand was confined to the deep extensors of the fingers and thumb and group of ulnar extensors, there being no movement of the upper arm and shoulder muscles.

At one time while examining the patient she had one of the above described convulsive attacks lasting two minutes. Consciousness was completely retained, and the movements were absent in all other parts of the body except in the arm and face as above mentioned. At the conclusion of this seizure the hand and face remained quiet for some time. These notes were made, October 1905, by Dr J. W. M'Connell, the assistant neurologist. Examination of the heart at this time showed the action of the heart to be irregularly intermittent; the pulse was small; a mitral systolic murmur was present. She was voiding very little urine.

On October 15 another examination was made, showing almost complete loss in convergence and in moving the eyes to the left and in associated upward movements, although this loss in associated movements was more marked on the right than on the left. The paralysis of the left upper extremity was deeper than when the previous note was made. No contracture was present, and the lower extremity was still not involved.

On October 16 the urine still continued to be scant, and she grew more apathetic and stuporous. The skin was clammy, and she could hardly be made to understand anything. Moist râles were distributed throughout the lungs. During the day she had six Jacksonian convulsive attacks like those already described. She died October 16, 1905.

Necropsy showed the heart to be very much enlarged, all the cavities containing post-mortem and some ante-mortem clots. The musculature of the ventricles was thickened. It was reddish pink in colour, was soft, flabby, easily pinched through,

and had a greasy surface. The mitral leaflet was thickened; the aortic leaflets were also thickened, especially the posterior. The aorta showed numerous patches of sclerosis, diffuse in character. Several ulcers were found in the abdominal aorta on its posterior wall.

The right lung was normal in size, the apex and anterior borders showing black pigmentation, in which areas the lung structure had a downy consistency. The left lung showed the same changes.

The spleen was small, and the capsule stripped easily and presented an iced-caked appearance. It cut with some resistance. The surfaces were dark red in colour and presented many islands of fibrous tissue.

The right and left suprarenal were normal.

The left kidney was normal in size, flabby, and cut with considerable resistance; the capsule was thickened and stripped with difficulty, leaving a granular surface. The cortex was narrow at points; medullary portion was purplish and congested. The pelvis contained an increased amount of fat.

The right kidney showed changes similar to those of the left. Cirrhosis of the liver was present. Examination of the stomach, intestines, pancreas, and other organs showed nothing abnormal.

On removing the calvarium, the meninges showed nothing noteworthy. A hæmorrhage was present in the right parieto-occipital fissure. A pathological area, apparently to the naked eye one of softening, was found in the precentral convolution in the arm area and upper portion of the face area (Fig. 1).

The ventricles were normal; the arteries at the base of the brain were hard and sclerotic.

A section of the pons made just cephalad of the fifth nerve showed an area possibly abnormal, this not including the motor bundles, but microscopical examinations failed to confirm this observation.

The specimens were sent to the Laboratory of Neuropathology at the University of Pennsylvania, where they were examined by Dr Wm. G. Spiller, to whom I am indebted for the following report:—

The middle third of the right precentral convolution is distinctly softer than the surrounding tissue, and on section the softening is seen to be the result of numerous hæmorrhages within

the cortex. A hæmorrhage of recent origin, about the size of a hickory nut, is in the outer portion of the right occipital lobe.

Microscopical sections through the affected precentral convolution show that the numerous minute hæmorrhages are almost confined to the cortex, although a few are in the subjacent white matter. At the region of greatest extension the anterior one-fifth of the precentral convolution alone escapes. The hæmorrhages extend deep into the central fissure, but do not pass beyond the bottom of the fissure into the post-central gyrus. The diseased cortex stands out very sharply to the naked eye. The transition from the normal to the hæmorrhagic cortex is quite sharp, even under the microscope. The pia adjacent to the affected cortex is also hæmorrhagic. A considerable amount of black granular pigment is found in the hæmorrhagic cortex. Numerous fatty granular cells and round cells are in the pia over the hæmorrhagic cortex. A small round hæmorrhage about ·5 cm. in diameter is found at the extreme lower end of the hæmorrhagic area. The sharp limitation to the precentral gyrus, and the almost complete limitation to the cortex of this gyrus and to its posterior half are very striking. There is no round-cell infiltration about the chiasm, but there is slight round-cell infiltration about the small vessels of the cerebral peduncles (Fig. 2).

The symptomatology and pathology of this case speak for themselves in the light of the short study of the literature of the subject prefixed to the history. The strict limitation of the lesion to the cortex anterior to the bottom of the central fissure is of interest in connection with the question of the absolute precentral position of the motor zone. The disturbances of ocular movements and the hemianopsia apparently present were probably due to the gross hæmorrhage in the occipital region.

EIGHT CASES OF HEREDITARY SPASTIC PARAPLEGIA.

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THE study of heredity in medicine is becoming increasingly important from both a theoretic and practical standpoint, and it is more and more apparent that many of the problems can be solved only after the accumulation of a large amount of suitable material. Particularly is this evident in the case of the affections that we may assume to be pure biological variations, such as occur especially in the nervous system, and it is therefore expedient that such cases, certainly those belonging to the rarer varieties, be recorded whenever an opportunity for investigating them presents itself. I have recently had occasion to examine a family in which eight cases of hereditary spastic paraplegia occurred in a single childship,[1] and wish here to publish a brief account of the family. The family, which has been under my observation for over a year, was shewn at a clinical meeting of the University College Hospital Medical Society in November 1905. I do not propose to refer to any of the extensive literature of the subject, as I hope to incorporate a discussion of it in a later contribution on an allied topic.

CLINICAL FEATURES.

As the features were so similar in the different members of the family, I will describe only one case in detail, and then mention the characteristics of the other members. It may be taken that the description of the first case applies to the others in all respects, except where the contrary is expressly stated.

[1] Term introduced by Professor Karl Pearson to signify the total offspring of a single union.

Ernest L., the second boy, aged 14,[1] is the most advanced case, and accordingly I select him as the type. He first came under my observation at the Old Kent Road Centre for Physically Defective Children, in November 1905. His mother had considered him normal in all respects until after he began to walk, which he did between the ages of one and two. Shortly after this she noticed that his gait was becoming stiff and clumsy; it was manifestly so by the age of three. About the same time she also began to notice that his feet were changing shape. The right foot was the first affected, but the left was implicated within a few months. The subsequent course of the disease was quite gradual and slowly progressive. Of late years, perhaps for three or four, the progress has ceased altogether, or has been so slight as to be inappreciable. Nothing else of importance was gleaned from the history. The boy thinks that he is, if anything, better than he was two years ago. He suffers from no symptoms beyond the difficulty in walking, which he describes accurately as a stiffness. He states that it is rather worse during the first few steps that he takes, particularly if he has been resting for a while; this, however, is certainly not pronounced, and I could not verify it objectively. He knows of no other circumstance, such as fatigue, time of day, etc., that in any way modified the symptom.

Examination.—The boy is intelligent and more advanced in his studies than is usual of boys in his station. His visceral organs are sound, and there are no indications of past syphilis. His cranial nerves, articulatory capacity, and sense organs are normal, as are his organic reflexes, and there are no sensory changes or defects in co-ordination. There are no trophic disturbances; the feet are bluish, but not cold.

Attitude.—Both feet are in a position of talipes equinovarus with pes cavus. This is much more pronounced on the right side. When standing he rests firmly on the left foot, but when doing so only the last two phalanges of the first two or three toes of the right side touch the ground; the right knee, and to a less extent the hip, is partly flexed and the thighs adducted. When he lies down the deformity is less, as it is also in a foot on which is placed the whole weight of the body; it can be made to disappear by passive manipulation. There is only very slight compensatory scoliosis, and no other deformity.

[1] The age and other points in the description are given as in January 1906.

Motor System.—The development of the limbs is good, except that the legs are thinner than normal; the right calf measures 21 centimetres at its maximum circumference, while the left measures 24. There is no local atrophy and no tremor of any kind. The lower limbs shew a slight diminution of power, but this would probably not have been called pathological had not other signs of disease been present; it is not confined to any particular group of muscles. Both lower limbs are markedly hypertonic, the right much more so than the left. The gait is obviously spastic, even approximating to a crossed-leg progression. The difficulty in walking is therefore made up mainly of two factors, the spasticity and the mechanical impediment due to the deformity of the feet and the contracture of the right lower extremity; it is only in slight part due to weakness. The muscular strength is very good elsewhere.

Reflexes. — The knee-jerks and Achilles reflexes are very markedly increased. Ankle clonus, of the organic variety, and patellar clonus are easily elicited. When one strikes the tendons on the dorsum of the foot, flexion of the toes, especially of the three middle ones, follows, Bechterew's sign being present. A crossed adductor-jerk is present. All these signs are more marked on the right side. The supinator and triceps-jerks are unduly active, as is the jaw-jerk, but the scapular-jerks were not obtained.

Babinski's extensor response occurs on both sides. The sign first described by Schäfer, called in America Gordon's paradoxical gastrocnemius phenomenon, is obtainable on both sides. Oppenheim's sign was not obtained, nor was White's. The cremasteric and abdominal reflexes are normal.

John, aged 17, is the eldest. Like all the other cases, he is not affected so severely as Ernest, and indeed is not prevented from earning his livelihood at various unskilled manual employments. His gait is, however, so spastic that he can move along the street only at a slow pace; he uses no support. The talipes is just present on the left side, but is very decided on the right; the increase in the deep reflexes is more pronounced on the right side. The jaw-jerk was not obtained, but the supinator reflexes are very active.

Arthur, aged 11, is affected to a considerably less degree than Ernest, and attends an ordinary elementary school in Mina Road.

There is slight talipes on the right side, none on the left. The other signs resemble those of John's, but the gait is decidedly better, so that he can join in the school games with fair capacity.

Mabel, aged 9, is the only girl, and is the only child that has escaped. Her reflexes are absolutely normal, and there are no evidences at all of any nervous affection.

The next two children, aged 7, are twins. Of these, William is affected decidedly more than Alfred. The result is that he, like Ernest, has to attend at the Centre for Physically Defective Children, while Alfred, like Arthur, goes to the ordinary school. William has talipes on both sides, but as it is not very advanced he can manage to hobble about pretty quickly, though not very securely. Babinski's sign is present on both sides, but the ankle clonus, the increase in the deep reflexes, and the talipes are all more marked on the right. The supinator-jerks are active, and the jaw-jerk can be elicited.

Alfred has only very slight deformity and spasticity. He can run about fairly well. The deep reflexes are obviously increased in the lower extremities, but not in the upper. Babinski's sign and ankle clonus are present; the jaw-jerk not so. The signs are all symmetrical.

Henry, aged 5, is affected to a moderate degree. He can walk fairly well. The supinator-jerks are active; the jaw-jerk was not obtained. The signs are symmetrical.

Sydney, aged 3, shews the following signs equally on the two sides. The lower limbs are slightly spastic, and there is a commencing talipes. The knee-jerks are markedly increased, as are the adductor- and supinator-jerks. The jaw-jerk was not obtained. Patellar clonus is easily evoked, but not ankle clonus. The plantar reflex is usually extensor in type, but sometimes flexor. The boy can walk fairly well, though his feet scrape the ground at times.

Albert, aged $1\frac{2}{12}$, is the youngest. He cannot walk or stand, but this is perhaps due to rickets, from which he suffers badly. It is difficult to estimate the tonus of the limbs, but they are certainly not rigid to any advanced degree. The knee-, supinator-, and adductor-jerks are all increased, and a crossed patellar reflex is easily obtained. A slight ankle clonus can be evoked. The

plantar reflex is extensor in type. There is no obvious talipes. The signs are symmetrical.

Summary of Cases.

The cardinal signs are spasticity of the lower limbs, talipes equinovarus, and changes in the reflexes indicative of an organic affection in the pyramidal tracts. It is a curious chance, and probably nothing more, that the signs are most marked on the right side in the four eldest patients, and symmetrical in the four youngest. Paresis is only of the slightest intensity. The cardinal signs are present to an unequal degree in the different cases, and this is in no way proportional to the age of the patients. The spasticity and talipes can be correlated in degree fairly well; they are present in all but the youngest. The changes in the reflexes form indisputable evidence of an organic pyramidal lesion. The arm-jerks are increased in all the cases, but in none is there any other evidence of the upper extremities being affected. The jaw-jerk was obtained in only two of the cases, the two worst. In no case are there any pseudo-bulbar symptoms present.

Family History.

For reasons indicated above, I think it worth while to give the family history in some detail, for one of the main obstacles to research on the types of heredity in such affections is due to the fact that writers so frequently content themselves with making negative statements. It is all-important to state precisely not only how many individuals *were* affected, but how many *were not* affected. All the individuals mentioned below are known positively not to have suffered from the affection, nor had any similar case been heard of by any member interrogated. The existence of the affection can therefore be excluded, with a fair probability, in the case of at least seventy members of the stock, including the members of the most important line for over 150 years.

Parents.—The parents are not related to each other. They are healthy, and give no evidence, either in the history or by

examination, of having had syphilis or any other relevant affection. They shew no abnormal physical signs in the nervous system. The mother has had no miscarriages, and none of her children have died. Her labours had progressed with moderate facility, instruments having been used in only two instances: even in these the children did not show any asphyxia or undue difficulty in starting respiration.

Mother's Relatives.— The non-affection of the only girl in the childship naturally led one to suspect that the malady might have been transmitted through the females. I therefore paid special attention to the maternal line, and the female part of this in particular. Mrs Leach was the eldest child, and had one sister and five brothers in that order. The sister died at 30, leaving an only child, who is now an unmarried girl of 19. The brothers are all married, and all, except the youngest, have children.

Mrs Leach's father died of apoplexy at 41. Her mother was ninth in a childship of eleven. Of these, the eldest, a male, died of cancer of the lip. The second, a male, died of apoplexy. The next four were all unmarried females; only the last of these survives, aged 81. The seventh, a male, died late in life, cause unknown. The eighth, a woman, was married, but died without having any children. The tenth, a woman, had as offspring only one girl, who died of pulmonary tuberculosis without marrying. The youngest, a man of 69, is alive, and has extreme scoliosis dating from infancy.

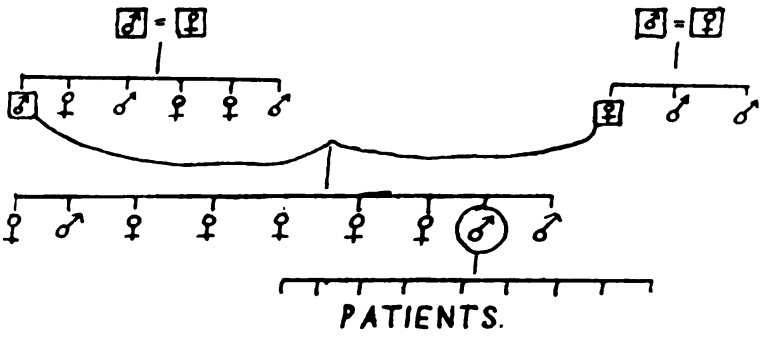
Mrs Leach's maternal grandfather died at 65. He had one sister and three brothers. Her maternal grandmother died at 67. She had one brother and one sister; the latter had one child (male), who died in infancy.

Father's Relatives. — Mr Leach was eighth in a childship of nine. They all married and had children except the fifth and sixth, both sisters. His mother was the eldest child; she had two brothers. His father was also the eldest of six children.

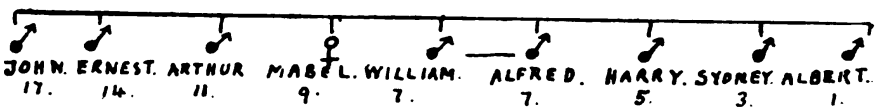
I append genealogical trees, so far as I could trace them. As regards the signs used therein, they are mostly those advocated by Mr Nettleship, and generally adopted. A wavy line = order of siblings[1] unknown. A bar indicates twins.

[1] Brothers and sisters (*Geschwister*) in the same childship.

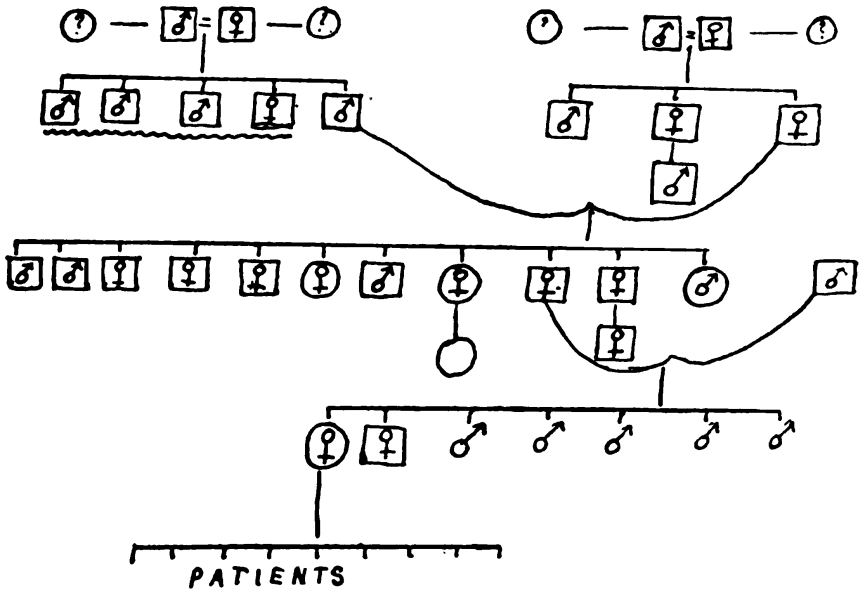
(?) = Number of siblings unknown. A circle = individual examined by the writer. A square = individual known to be dead. Present cases are indicated by dark shading.



PATERNAL SIDE OF FAMILY



CHILDSHIP NOW. REPORTED.



MATERNAL SIDE OF FAMILY.

Diagnosis.

It is evident that we have to do with a diffuse affection of the pyramidal tracts, and, so far as the signs point, of them only. There is no indication of the presence of any local lesion. The three conditions chiefly to be considered are hereditary spastic paraplegia, transmitted syphilis, and Little's disease. Of these, syphilis can certainly be excluded, for there we know of no form of the disease that will produce a progressive diffuse lesion of the pyramidal tracts at this age without giving rise to any other indication of its presence in either the parents or children. The freedom of the girl from disease is almost certainly more than a coincidence in a family of this size. As to Little's disease, the facts that intelligence is unimpaired, that there is no strabismus, fits, or any other indication of intracranial affection, are all presumptive evidence against this diagnosis. There are, however, rarer cases of Little's disease in which the effects of the cortical lesions are manifest only in the lower extremities. But it is most unlikely that this would happen in eight children out of nine in one family, and especially when there is none of the characteristic history indicating this cause. Further, the age at onset, and the peculiar mode of progress of the condition, in no way tallies with this supposition.

In support of the diagnosis implied in the title to this paper, the following reasons may be adduced: affection of all the male children, the only female escaping; absence of any assignable cause; first evidences of the malady between the ages of one and two, this being so in all the cases; striking similarity of all the cases to one another, the only differences being those of degree; similar course in all, namely, a very slowly progressive one, never reaching a condition of total incapacity; close resemblance in every detail to published cases which proved in fact to be transmissible through several generations.

I use the word "hereditary" in preference to "family," because—granted the correctness of the diagnosis—the condition is hereditary in the scientific sense; that is, it is due to an innate biological variation capable of transmission through a number of generations. In medical writings the word "hereditary" is often employed very loosely, for instance in the phrase "hereditary syphilis"; in this instance it is used to denote a

condition that is not an inborn variation, and therefore not one transmissible through several generations. It might be preferable to employ the word "family" to describe such a condition, and others that may occur in different members of the same family, due to a common extrinsic cause usually infective in nature. It seems illogical to separate a class of case, such as that described in the present paper, from that in which cases are observed in another generation as well, calling the one "family" and the other "hereditary." Such a distinction is practically meaningless, for it depends only on the extent to which the other generations are searched. Mr Nettleship has shewn clearly that the more extensive this search, the greater the number of cases that pass over from the family to the hereditary group, without of course there being any change whatever in the nature of the cases. That long intervals, even extending over several generations, may occur between the outcropping of such variations is of course in accord with what we know from biology of such variations. In the cases recorded in this paper, the original diagnosis would be only confirmed by the discovery, on the one hand, of additional cases in remote ancestors, or by the event, on the other hand, of similar cases occurring in the descendants—a fact that might be read as indicating that the present cases were the starting-point of the inborn variation in the stock.

AN INVESTIGATION INTO THE ARRANGEMENT OF THE ACHROMATIC SUBSTANCE OF NERVE CELLS, AND OF THE CHANGES WHICH IT UNDERGOES IN VARIOUS FORMS OF MENTAL DISEASES.[1]

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Introduction.

The present investigation was primarily undertaken with the intention of demonstrating the changes in the achromatic structure of the nerve-cells in pathological conditions. Two methods were principally used as representing two schools of teaching, viz. Bethe's original and Lugaro's colloidal silver method. The former shows the achromatin arranged as fibrils streaming through the cell, on the appearance of which the author has based his theories of nerve-cell relationship and conduction; the latter shows the substance arranged as an interlacing meshwork in the cells, and it is claimed by its author to be a much truer representation of the actual arrangement. It was found necessary to undertake an investigation into the arrangement of the structure in healthy states, and brain tissue from animals was used for the purpose. Other methods were also sometimes tried, viz. Ramon-y-Cajal's No. 3 method, and the pyridin methods of Donaggio for the demonstration of the endo- and pericellular structure.

Intracellular Fibrillary Arrangement.

In brief, Bethe originally claimed for the larger cells that a certain number of fibrils run through the apical process, cell body, and basal dendrites, a few pass from dendrite to dendrite; the fibrils are most numerous in the apical process, less numerous in the cell body, and fewest in the dendrites. The majority of the fibrils from the apical process form a reticulum round the nucleus—the deep or perinuclear reticulum. More superficially the fibrils are independent, and pursue a free course through

[1] Part of a thesis submitted for the degree of M.D.

the cell. The axis-cylinder arises from the perinuclear reticulum, which becomes arranged into independent fibrils, much finer in calibre than those of the dendrites; it also contains a certain number of coarser dendritic fibrillæ.

The independence of the fibrils was criticised by Donaggio, who maintained that they form a network within the cell, denser round the nucleus, and showing more open meshwork at the periphery. His conclusions in the main were supported by Rossi, Soukhanoff, Ramon-y-Cajal, Weiss, Azoulay, and Marinesco. Towards the end of 1905, Donaggio published the results obtained by his pyridin and thionin methods. He alters his position somewhat, and describes two kinds of cells: (1) Those with endocellular network only; (2) those with endocellular reticulum and fibrils coursing through the cell, and maintaining their individuality. He declines to express an opinion with regard to the anastomosis of these long fibrils; he believes the methods of Ramon-y-Cajal to be unsuitable for the demonstration of the very fine fibrils. He describes various condensations of the endocellular network, in addition to the perinuclear ring. With regard to the origin of the axis-cylinder, he describes four different positions in the cell from which it may arise: (1) From the periphery of the cell reticulum. (2) From the reticular thickening which is not perinuclear. (3) From the deeper portions of the cells and fibrils from the level of the nucleus. (4) Reticulum round the centre of the cell.

Rossi, Michote, and Lugaro believe there are no long fibrils; the latter by his colloidal silver method says that there is anastomosis of the fibrillæ throughout the cell, and also in the axis-cylinder, that lozenge-shaped spaces are thus formed which give the appearance of fibrillæ when appressed.

Founded on the results of his personal observations, Donaggio puts forward a theory regarding the function of the differently constituted nerve-cells, viz.: in cells with endocellular network only, impulses are conducted only in one direction, and the reticulum is an apparatus for the reception and synthesis of stimuli transmitted by cellulipetal paths. In cells with two systems of fibrils, the long fibrils pass through the axis-cylinder, and might conduct impulses cellulifugally, whilst the reticulum within the cell receives the cellulipetal impulses.

Personal Investigations.

The material used was obtained from the ox, sheep, pig, and cat. The appearances presented by the cells from various parts differ considerably in the fibrillar and reticular arrangement. The ganglionic cells of the cortex have a much greater fibrillated structure than those of the medulla and cord. The arrangement seen in these cells differs very much from that met with in the smaller cells. By Bethe's method the larger fibrils are distinctly shown, and in some cases there is a partial staining of the finer threads, but the general appearance is such as to confirm the opinion that the staining process is incomplete. The large cells of the spinal cord do not show the apparently independent fibrils, but in their processes the appearances are similar to those met with in the cortical cells.

The tissues prepared by the method of Lugaro show the achromatic structure arranged as an interlacing network, with no independent fibrils, and greater detail of structure is more clearly brought out. The results obtained by this method also vary, especially in specimens obtained from animals. The tissues were placed in the fixing agent immediately after the animal was killed; tissues from the human subject where the results were uniform were not obtained until a considerable period after death. The want of uniformity might be due to the action of the fixing agent on tissues before actual cell death had occurred.

The small and large pyramidal cells present a simpler endocellular arrangement than the layer of giant cells. The anastomosing reticulum is more distinct, less elaborate, and the meshes are fewer in number. With regard to the appearance of the meshwork, the results obtained confirm those of Lugaro. The fibrillation seen in the apical process is only apparent, and is made up by the apposition of numerous diamond-shaped meshes of the reticulum; in the cell body they expand and become more open at the periphery of the cell, are smaller in the intermediate zone, and in the zone immediately round the nucleus they are smallest.

At the origin of the axis-cylinder there appears to be no actual rearrangement of the structures within the cell. The endocellular network seems to be wholly prolonged into the axis-cylinder sheath and is consequently appressed; this causes a

diminution in size of the reticular spaces at the cone of origin of the axis-cylinder. The cone itself is formed by the apposition of the meshes; this gives an appearance of a rearrangement having taken place, and also of long fibrils, especially at the periphery of the process. The cone may reach the perinuclear ring, especially in the human subject, where the endocellular network is more profuse and the axis-cylinder origin is consequently greater. In diseased states the connection with the perinuclear ring is the last to be destroyed. The dendrites can be followed through the pericellular nets to the peripheral zone.

The cells of the medulla and cord very much resemble each other, the three-layered arrangement of the intracellular network being very distinct. Donaggio has described various condensations of the endocellular network besides the perinuclear ring, but these condensations are very variable in their position and arrangement and might result from the sudden transition between the meshes of the different layers. The arrangement of the fibrils in the processes is shown by Lugaro's method to be a network. Bethe's method fails to reveal the secondary fibrils which complete the network; the interfibrillar substance is for the most part homogeneous.

Applied to the cerebellar cells the method of Lugaro has not been successful. The wealth of connections of Purkinje's cells is not seen; the basket arrangements round each cell are also very faintly indicated. This may be due to the latter structures being pericellular aud probably non-nervous.

Pericellular Network.

Three views are held with regard to its composition. It is—(1) a nervous structure; (2) neuroglial; (3) an artefact. Golgi and Martinotti first described it as neurokeratin, and the latter looked upon it as an isolating body. Bethe found that his method stained it, and he believed the reticulum to be formed by the axis-cylinders of nerve cells and that it establishes continuations with the neurofibrils of the cytoplasm. Ramon-y-Cajal thinks it is not a real pericellular structure; Lugaro supports this and thinks the appearances are due to the coarser meshes of the superficial layers of the spongioplasm. Marinesco thinks the appearances are artificial. Van Gehuchten thinks the

presence of a network is doubtful, but it is not a conducting body. Perhaps the view advanced by Donaggio that the plexus is a demonstrable structure, that it is in close relationship to the nerve cells, but that it is of neuroglial origin and consequently has nothing to do with nerve cell function as that is usually understood, is the theory that finds most acceptance at the present time. In 1896 he described structures which he maintained were similar to those described by Nissl, Ramon-y-Cajal, Semi Meyer, Aldren Turner, and Hunter, and which he believes to be neuroglial in origin. Further papers in 1904 and 1905 serve to confirm him in his belief. Held, who formerly believed the structures to be nervous, now supports the view that they are neuroglial. In the meshes of the pericellular reticulum are minute ray-like structures which stain by Donaggio's method and which are composed of a single centre which does not stain and numerous fibrils radiating out from it. "Radiations" or "Raggiere" is the term given them by their author. Held confirms the presence of the radiations, and thinks they are the same structures described by him under the name "Sternformigen Haufen"; he thinks they are non-nervous, but Donaggio does not give an opinion. Ramon-y-Cajal suggests that the central unstained portions correspond to the "Buttons of Auerbach," which are impregnated by his method but are not coloured by Donaggio's process. The embryological researches of Fragnito, Capobianco, Paladino, and Seniti support Donaggio's view of the neuroglial origin.

Do Nerve Cell Fibres Anastomose?

Golgi thought that the axis-cylinder broke up into a network round the adjacent nerve cells; Vassale and Donaggio suggested there might be an anastomosis between the gemmulæ on the protoplasmic prolongations of one cell and the axis-cylinder of another. In the cerebellum Hill thought it possible that the axis-cylinders of the granule cells ended in the gemmulæ of the processes of Purkinje's cells. Held believes in the continuity of the axis-cylinder and adjacent cell processes. In invertebrates Apathy, by means of his neurofibril method, demonstrated that primitive fibrils may run through several ganglion cells and form numerous anastomoses; he thinks the whole mass of nerve

fibres is one continuous sheet of connections. Bethe supports Apathy's views of anastomosis in the intercellular reticulum. Ramon-y-Cajal does not believe there is continuity, but considers that there is contiguity between the nerve cells and fibres, and that the chalices described by Held envelop but do not penetrate the cells. Veratti, however, regards these chalices as products of partial impregnation of a membrane which surrounds unipolar cells. By means of his newer processes, Ramon-y-Cajal has described structures on the cell body which he terms "Buttons of Auerbach"; they are black, often granular, the centre is sometimes clear, they are very numerous on the cell bodies and processes of the cells of the spinal cord and bulb; they have not been demonstrated in the cells of the cerebral cortex. Van Gehuchten thinks that these buttons end independently on the cell body, that they do not form a network, and that they are the same as the structures described by Held and termed by him "Endfusse." The suggested relationship between these and the "Radiations" of Donaggio has already been mentioned.

With regard to the nerve fibre terminations in the cells of the acoustic centre, it was thought by Held that the pericellular baskets which enveloped these cells were formed by adjacent nerve fibres, but in 1903 Donaggio was able to show that the axis-cylinders and the large nerve fibrils are distinct; he believes that the acoustic terminations of Held are not really nerve fibre terminations, but are part of a fibrillar conducting apparatus.

That specialised pericellular structures exist, must, I think, be allowed, at any rate in the larger cells of the cerebral cortex and in those of the medulla and cord. The colloidal silver method shows differentiated structures in each of these localities; the cells show a reticulum, the meshes of which are more rounded and the fibrils are of finer calibre; it appears in continuation with the cell body, is prolonged on to the dendrites and axis-cylinder, and is seen as a rudimentary web at the origin of the processes. The boundary of the cell is marked by a fairly regular line, on the outer side of which is the covering described. Further, the dendrites are sometimes seen passing out from the boundary of the cell and covered by a sheath, in the same way that the spinal nerves emerging from the cord carry a covering of the meninges. Adjacent cell processes can

be traced on to this net, but it is not determined whether there is actual fusing or not. Regarding the nature of these structures, they are probably different in constitution to the nerve-cells, and are probably of the nature of specialised neuroglia. In this connection the findings of Donaggio are of the highest importance, and point to an ultimate solution of the difficulty.

Development of Nerve-Cells and Fibres.

Before considering the functional activity of the nerve-cell, it is necessary to review the work that has been done of recent years on the development of the nerve-cell and its fibres. Up to recent years the theory that each nerve-cell and its fibres was developed from a single unit was generally accepted. During the past six years or so, Fragnito and others have been endeavouring to disprove this. In the chick he shows the epithelial and the germinal cells to be quite distinct. The former form the neurospongium—a substance of great tenuity, which contains blood and lymph vessels. Neuroblasts appear later as deeply staining, round or oval bodies, which tend to form groups, and to wander away from the neural tube. Each group is composed of: (*a*) primary neuroblasts—may be one or more—which form the nucleus of the adult cell; (*b*) secondary neuroblasts—several in number—which form the various parts of the cell protoplasm. There is no karyokinesis. Later the secondary neuroblasts lose their contour, fuse together surrounding the primary neuroblasts, and so completing the appearance of the adult cell. Capobianco confirms these findings; he compares the number of nerve-cells formed with the number of neuroblasts, and he finds that in two cases the proportions were 1 to 2·83 and 1 to 3·02. These findings lend considerable support to the theory.

In 1903 Fragnito also dealt with the development of the peripheral nerves, and sought to show that they were developed from rows of cells which gave rise to two processes. These processes unite with similar ones from other cells, so that a beaded fibril results—the beaded appearance being due to the persistence of the cell. By the coalescence of a number of fibrils a fibre is formed; the axis-cylinder and dendrites grow as distinct structures after this manner, and when complete, one

end becomes fused with the secondary neuroblasts at the edge of the cell body.

Of late years also much attention has been directed to regeneration of nerves after injury. The older theory of Wallerian degeneration and regeneration from the central end of the divided nerve has been severely criticised. The peripheral theory of regeneration was started by Büngner in 1891, and has since been strongly advocated by many writers. Degeneration begins in the peripheral end in a few hours, and is complete in three to four weeks. The nuclei of the neurolemma sheath take on the functions of neuroblasts, become arranged in rows, and in course of time give rise to axis-cylinder and myelin sheaths. Bethe claims to have demonstrated this experimentally, but his results were adversely criticised by Münzer. Fleming thinks that both central and peripheral regeneration occurs. Cases of rapid reappearance of sensation (one to two days) after accident is attributed to nerve-twig anastomosis, or to peripheral regeneration.

Considerations on the Neuron Theory.

Since the theory of the pluricellular origin of nerves has received so much attention of late years, the neuron theory which is built on the single cell theory has been vigorously attacked. Golgi never accepted the theory, and was the chief critic between 1891 and 1897. His criticisms were supported by Apathy's researches on invertebrates, and Bethe's work on vertebrates was similarly directed; the latter observer endeavoured to show that nerve function took place in the intercellular network where the neurofibrils anastomosed; he attempted to support his conclusions by experiments, but these were not conclusive. In 1898 and 1899 Lugaro attacked the critics, and foreshadowed a view which he later laid more stress upon, viz. that mere continuity of nerve-fibrils would not necessitate the destruction of the neuron theory; that were Bethe able to conclusively prove that acts took place in the intercellular network, and that the nerve-cells only exercised a trophic function, it would not seriously interfere with the theory, in so far as it affirms an embryological and trophic autonomy. In invertebrates Lugaro suggests that a fibrillar anastomosis is com-

patible with the performance of simple acts, but for finer nervous interworkings, as in the higher animals, it is more easy to establish associations in complex acts by simple contiguity. Van Gehuchten, Barker, and Ford Robertson support Lugaro's reasoning.

Since 1900, Fragnito and others, basing their conclusions on the pluricellular origin of nerves, have added more criticisms against the theory. Durante, in the course of his criticisms, says that he believes that the fibres are not merely inert conductors, but that each segment takes part in the conduction of the impulse, and the relative slowness of nerve transmission is due to the time occupied between the neuroblasts, each of which is active in the process. In 1904 Bethe states the arguments for and against the theory, and remains unchanged in his position. About the same time Lugaro elaborates the views he expressed in 1899, and points out that the anatomical unity of the neuron is conceivable in two senses, viz. (*a*) in a narrower sense implying a cellular unity, (*b*) in a wider sense as an "organic individuality," as a complete organ, leaving unprejudiced the question of its embryological origin and of its cellular "unity."

In examining the evidence that has been brought forward against the neuron theory, it is seen that most of the criticism is founded upon results obtained by Bethe's method. The author himself is the most zealous critic, but even he has been unable to demonstrate conclusively, either histologically or from experimental research, that there is a true anastomosis between the processes of one cell and those of another. The method itself is unreliable and inadequate to show the reticular arrangement. With regard to impregnation by silver nitrate, it is scarcely possible to draw conclusions from the results. It would be better to continue to regard it only as a useful adjunct to anatomical study. The further elucidation of the relationships of the pericellular nets and "radiations" of Donaggio will probably help to throw further light on the mode of nerve-cell termination. It seems to me that the modifications which Lugaro suggests, viz. each cell as an "organic individuality," can scarcely be permitted if the neuron theory is to be maintained. Relationship by contiguity of cell to cell is the main argument for the theory; and if continuity can be proved, it necessarily follows that nervous

phenomena must be explained on a different conception of cell arrangement. Besides, it is not quite clear what Lugaro means by "organic individuality," or how he proposes to correlate it with continuity of fibres. The embryological studies of Fragnito, Capobianco, and others, and the accumulating evidence of peripheral regeneration of nerve fibres, are certainly very suggestive; but on a matter of such moment, and one so full of debate, there has been too little confirmation of the findings, and consequently it must still be held that the neuron theory is the best suited to explain the various nervous acts.

Pathological Changes in the Neurofibrillar Elements of Nerve-Cells.

During the past six years various experimental researches have been carried out to demonstrate changes in the achromatic substance. These researches comprise results from four varieties of experiment, viz. cutting, evulsion, prolonged anæmia, and the toxins from tetanus and rabies. Three constant changes were noted, viz. (1) reaction, (2) degeneration, (3) reparation. The more severe the lesion, the more intense are the cell and fibre changes, but these changes are practically the same whatever be the lesion. The cells swell, then shrivel, nucleus is displaced, chromatolysis, loss of striation, powdery appearance of the chromatic elements; the changes spread from the cell body to the fibres; the reticulum disappears.

Marinesco has described regeneration of the achromatic structure after injury. The generated material differs from the original in that the strands are thicker, more opaque, and hypertrophied.

In various diseased states, *e.g.* myelitis, meningitis, and brain softenings, the changes have been described by Marinesco. Chromatolysis and breaking up of the fibrils go on side by side: they may commence either at the centre of the cell, at the periphery, or diffusely.

The following notes are an epitome of an investigation into the changes met with in the achromatic substance of nerve-cells in about thirty cases of the insane. The specimens were routinely collected from the cases in the post-mortem room, and practically nothing was known about them until the microscopical examination

was finished. The precentral convolution was always examined, in many cases also parts of the medulla, cord, and cerebellum.

Group I.—Six cases. *General Paralysis.* Small pyramidal cells much reduced in number, and staining reaction-cell spaces frequently contain only traces of amorphous material. Large pyramidal layer—the cells are more distinct; fibrillation and pigmentation, especially at the base of cell, pronounced. The ganglion layer shows the cells much swollen and globose, or shrunken, their processes broken off; the pericellular net as a ragged covering; unequal staining of the cell body; endocellular reticulum altered; the various condensations absent; the meshes larger, and their outline fainter. As is usual, much cell pigment is present, and is contained in meshes larger, more rounded, and coarser than those usual to the cell. Figs. 4, 5, 6, 8.

Group II.—Organic Dementia, in which cerebral softening has occurred. The general character of the changes resemble those met with in the former group, but the cell disintegration is greater; the brain tissue was softer, peri- and endarteritis were pronounced—small pyramidal cells very fragmentary—the cell outline of the large pyramids was very faint; many large neuroglia-cells seen packed between the nerve-cells — axis-cylinders varicosed, thickened, and irregular—the nucleoli of the ganglion cell layer deeply stained, in contrast to the faintly-coloured nuclei and cytoplasm—pigment much increased —Purkinje's cells faintly coloured—the cells of the medulla and cord are in a state of very advanced degeneration, so that almost no healthy structure was seen. The cell processes and endocellular network seemed almost completely destroyed. For some time before death the patients were bedridden and helpless. Figs. 7, 9, 10.

Group III.—Dementia associated with various physical disorders—chronic interstitial nephritis—tuberculosis of kidneys and lungs. The intracellular structure of the small pyramids was not so disorganised as in the other cases; fibrillation persisted in the processes—the cells of the large pyramids were fairly numerous, but unevenly stained, the nuclei were usually displaced—the axis-cylinders were often well shown—pigment fairly large in amount—remains of perinuclear ring present—pericellular reticulum fairly well shown—interfibrillar spaces larger. Fig. 11.

Group IV.—Contains a series of six cases of *Chronic Brain Atrophy.* Great diminution in size and number of small pyramids, and also much alteration in shape—large pyramids similarly affected—endocellular network indistinct, fragmented, and granular—ganglionic cells similarly affected, and contain much greenish coloured pigment.

Group V.—Two cases of *Epileptic Dementia.* The structure of the small and large pyramidal cells is more profoundly affected than in any other condition examined—there is no trace of reticulum in the cells of either layer—the ganglionic cells are much altered. The medullar and nuclear are merely outlined. In the cerebellum the cells of the granule and molecular layers appear to be numerically less.

Group VI.—Includes one case of *Diabetes Mellitus* associated with *Melancholia.* The chief point of interest in this case is the want of staining of all the cells in the cortex, medulla, and cord, whilst those in the cerebellum were only faintly coloured. The intercellular substance was beautifully coloured, and by Lugaro's method the sections had the appearance of Weigert-Pal preparations; the cell spaces were empty, or contained only a little debris. Fresh collargol and control sections which stained well were tried, but with a negative result. The patient had suffered from diabetes for a number of years, and died of diabetic coma. The pancreas was much diseased. Fig. 12.

Group VII.—Chronic Mania associated with *Tuberculosis and Fatty Heart*—three cases. The percentage of stained cells in the small and large pyramidal layers is much larger than in other groups—the appearance suggests that the stage of disintegration is less advanced here. Although the ganglion cells present the usual features of disintegration, it was noted at the time of examination, and before the mental state was known, that the changes were not so advanced. In the cerebellum the intercellular substance of the granule layer is increased.

As a result of these observations it is seen that the achromatic structure of the cells is profoundly affected in mental diseases, and that the changes differ chiefly in degree rather than in kind in the various conditions. The case of diabetes mellitus presents very unusual features, but as it is a single case, and so

lacks confirmation, too much stress is not laid upon it, and it can only be regarded as an interesting condition.

Much has been written about the significance of pigment in nerve-cells, as its presence is such a constant feature in diseased conditions. It is most frequently found at the part of the cell where there is supposed to be greatest functional activity, although this is not always so. It is contained in a reticulum which differs from that of the cell proper, in that the meshes are larger, more irregular, and the fibres which form the meshwork thicker. Marinesco thinks that the pigmentary reticulum is distinct from that of the cell, and the appearances presented by the specimens under examination go to confirm that view. The fact that it tends to increase in old age, and in the more chronic diseased mental states associated practically constantly with disease of the blood-vessels, implies a gradual interference with the normal nutritional supply to the tissues; and it is easily conceivable that such a reduction of nutrition would result in diminished metabolism within the cells, and so to an accumulation of partially reduced effete bodies which appear as yellow pigment.

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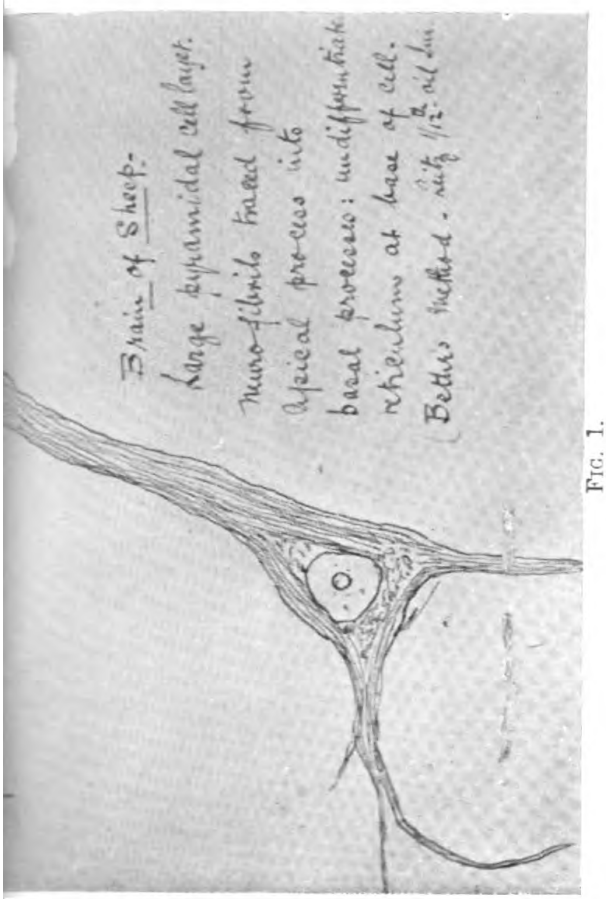

FIG. 1.

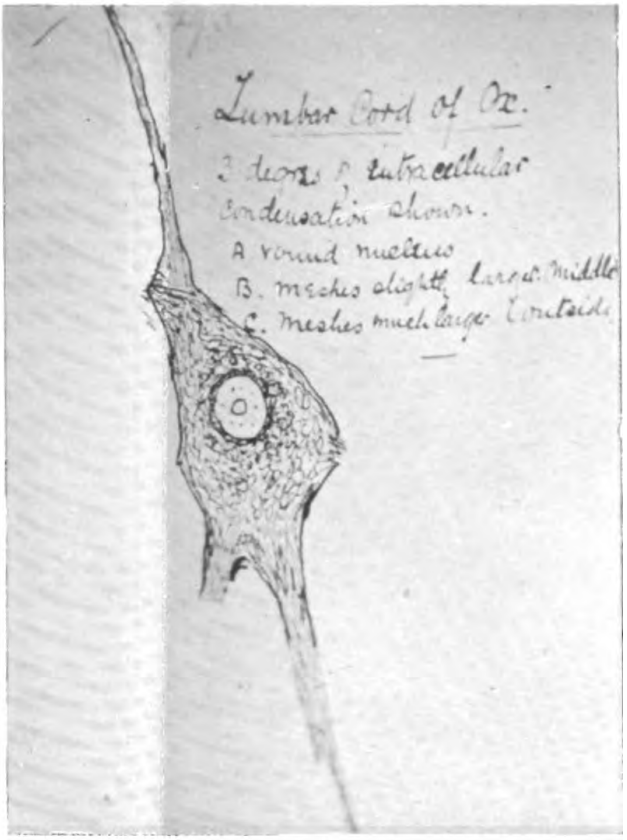

FIG. 2.

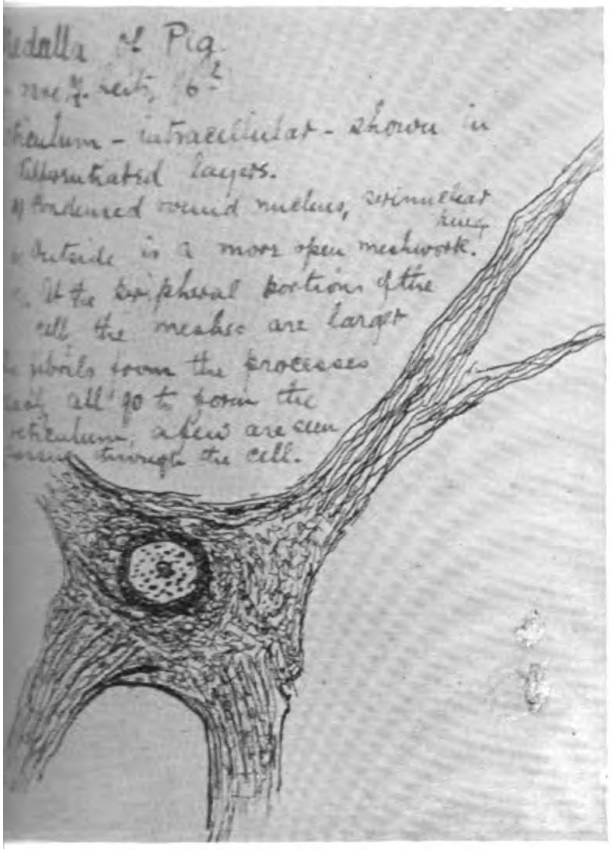
Medulla of Pig.
reticulum - intracellular - shown in
differentiated layers.
condensed round nucleus, reticular
outside is a more open meshwork.
In the peripheral portion of the
cell the meshes are larger
fibrils from the processes
all go to form the
reticulum, holes are seen
through the cell.

FIG. 3.

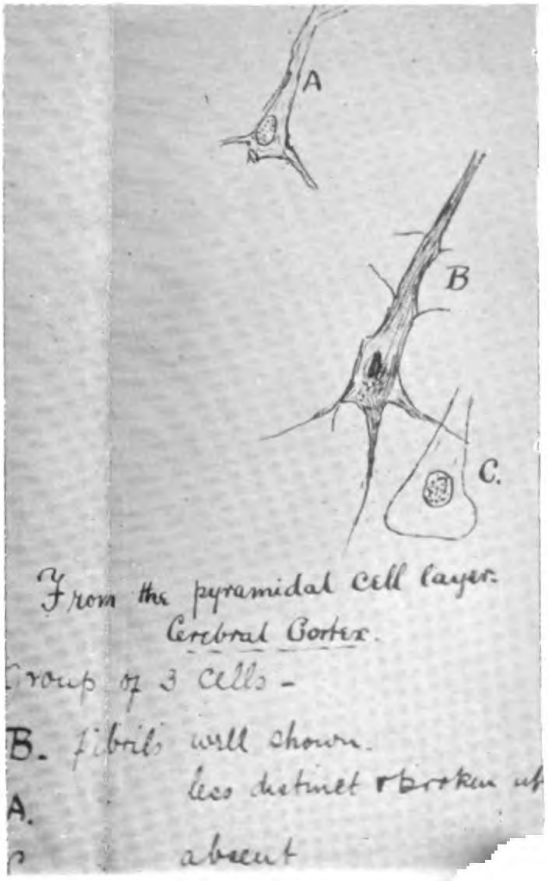

FIG. 4.

FIG. 3. Medulla of pig—large cell shows anastomosing reticulum in processes; three layers of cell condensation; prominence of perinuclear network; appearance of independent fibrils which are really anastomosing network; larger meshes at origin of axis-cylinder. (Lugaro.)

FIG. 4. Male G.P. group of three cells from large pyramidal layer, showing inequality of staining. (Bethe—toluidin blue.)

FIG. 5. Female G.P. group of three cells from ganglion layer; globose form; internal reticulum much altered in form. (Bethe.)

FIG. 6. Group of cells from the ganglion layer, motor cortex of a male G.P.; extensive pigmentation is seen in all the cells; endocellular reticulum is fairly represented. (Lugaro.)

FIG. 7. Male—Organic dementia—groups of three cells from ganglion layer; they are much swollen, extensively pigmented, distorted; internal reticulum faint and much altered. (Leitz, ½ O. I. Bethe.)

FIG. 8. Female G.P. cell in an early stage of degeneration, surrounded closely by connective tissue cells. ? Phagocytes. (Bethe.)

FIG. 9. Male—Organic dementia—cerebral softening—axis-cylinders showing variations in size and varicosity. (Bethe.)

FIG. 10. Female—chronic mania—cerebral softening—extensive pigmentation of ganglion cell, which is cut obliquely; fibres composed of reticulum. (Leitz, ½ O. I. Lugaro.)

FIG. 11. Male—dementia following delusional insanity. Tuberculous kidney. Both cells show well-stained internal reticulum. A shows pericellular network; B shows a peculiar elongation and pigmentation at the base of the cell. (Leitz, ½ O. I. Lugaro.)

FIG. 12. Male — diabetes mellitus — melancholia — low power drawing of portion of cortex, showing absence of cell staining. (Lugaro.)

Abstracts

ANATOMY.

CONTRIBUTIONS TO THE HISTOLOGICAL LOCALISATION OF
(48) **THE CEREBRAL CORTEX. FIFTH COMMUNICATION.** **(Beiträge zur histologischen Lokalisation der Grosshirnrinde. Fünfte Mittheilung.)** K. BRODMANN, *Journ. f. Psychol. u. Neurol.*, Bd. 6, 1906, S. 276.

THE paper under review is Brodmann's fifth contribution to the architecture of the cerebral cortex. The investigation is based on the study of the cell arrangement alone. The aim of the present contribution has been to ascertain how far the cortical structural areas which exist in man can be recognised in the lower mammals, and secondly to contribute to the discussion of the homologies of sulci.

Only a small proportion of the interesting facts brought to light in the present paper can be referred to in an abstract; the general results are similar to those made familiar to English readers by Campbell's publications, but the original article must be consulted by those interested in study of the brain, especially with regard to the localisation of function in the cortex.

Brodmann's description of the lamination of the cortex differs from Campbell's in that he recognises only six instead of seven layers. His layer (1) is the lamina zonalis, which is devoid of cells; (2) includes the plexiform and small pyramidal cell layers of Campbell; (3) the medium-sized and large pyramidal cells; (4) the internal granular or stellate layer; (5) the layer of ganglionic or deep pyramidal cells; and (6) the deep spindle-shaped or polymorphous cells. Even in regions where one or other of these layers has disappeared, or where two or more have fused together, or where they are apparently increased in number by subdivision, the six-layer type is always recognisable during some period in the course of development. This deviation from the type arrangement in the adult condition is well illustrated by the two structural areas which have been mapped out by the author in representatives of practically all the mammalian classes. The area gigantopyramidalis (precentral type of Campbell) is distinguished by the great reduction or absence of the fourth layer, though it is well marked in it during fœtal life; while the

calcarine type (area striata of Elliot Smith, visuo-sensory of Campbell), which is distinguished by a doubling of the fourth layer and the appearance of gennaris layer (lamina granularis intermedia) between the subdivisions, making the cortex appear eight-layered, has the typical six-layer type in the sixth fœtal month.

Many of the cortical types are more difficult to recognise and less typical in some of the lower mammals; for instance, in many of the lower orders the fourth layer is not divided up, *i.e.* there is no gennaris layer in the homologue of the area striata. Homologous cortical fields may be consequently dissimilar in structure.

Area striata.—The topography and relations of this area in man is almost identical with the distribution of Campbell's visuo-sensory type of cortex. From his earlier investigations of the European brain, the author was unable to accept Elliot Smith's statement that in many brains (those of Egyptians and Sudanese were studied by E. S.) the area striata extends on the lateral surface of the hemisphere almost up to the sulcus simialis (S. lunatus of E. S.—the homologue of the Affenspalte). But on examining Javanese brains it was found that Elliot Smith's observations on the Egyptian's held here too, that the area striata extends on the lateral surface almost as far as the S. simialis, so that from the similarity to the condition found in the higher apes it must be admitted that this fissure is the homologue of the Affenspalte.

In the higher apes (Orang) the extent of cortex occupied by the area striata on the mesial and lateral surfaces of the occipital lobe is about equal. The dorsal and ventral borders of the area striata are not limited on the lateral surface by the S. simialis and S. temporo-occipitalis lateralis, as has been asserted (Schlapp); in none of the apes does the area extend as far as the furrows, but the space by which it falls short of them is variable in different species. In the orang the area striata occupies practically the whole cuneus.

In the lower apes the area striata is relatively much larger than in man, and the greater portion of it lies in the lateral surface of the hemisphere. Whilst in man, too, the area extends to the surface of the gyri for almost the whole length of the calcarine fissure, in the lower apes it lies buried for a considerable length of the oral part of the fissure, in many species the whole distance oral to the junction of its branches (involution, E. Smith), so that on the surface it is visible only in the caudal ends of the cuneus and lingual gyrus. There is not, however, "complete involution" of the area striata on the mesial surface of the occipital lobe in any of the apes, so Elliot Smith is incorrect in regarding the S. calcarinus proprius of men alone as the homologue of the calcarine fissure of the apes, and in refusing to admit an homology between

the human cuneus and that of the monkey, on the ground that part of the former is striate and the whole of the latter non-striate.

In the lemur class the distribution of the area striata on the lateral surface is practically identical with that in the lower apes, but on the mesial surface it occupies the greater part of the cuneus and the posterior end of the lingual lobe, in this respect more closely resembling the anthropoid apes and man. Unlike the arrangement in the primates, the oral end of the area is not buried in the calcarine fissure, but extends on to the surface of the lingual lobe.

In the cheiroptera the area striata loses the topographical relations to the sulci of the occipital lobe which have been described in the higher orders. Owing to the great development of the rhinencephalon, it comes to lie on the dorsal margin of the posterior portion of the hemisphere.

The anatomical distribution of the area striata in the carnivora is of special interest, as Munk and Berger have localised the visual centre by experiment in the dog and cat in the dorso-caudal part of the lateral surface of the brain. B. has already pointed out that the centres outlined by these physiologists do not correspond to the area striata as determined by histological structure, and in this he is in full agreement with Campbell. The area striata occupies the caudal two-thirds of the lateral and medial surfaces of the gyrus marginalis. On the median surface the S. post- and supra-splenialis lie within it, and are probably the homologues of the S. retro-calcarinus (E. Smith). The area striata is bounded in part at least by the S. lateralis and splenialis; it does not at all correspond to Munk's visual centre, and the latter does not coincide with any structural area.

In the rodents and insectivora the area striata lies on the dorso-caudal surface of the hemisphere; it is more extensive laterally than mesially. There is no fissure which can be regarded as the homologue of the calcarine. In these orders the cortical structure is peculiar; it is relatively very broad owing to the great development of layers v. and vi., but layers ii. and iv. are very narrow.

Köppen and Löwenstein have described the gyrus splenialis as the homologue of the area striata in Ungulates. B. has been unable to find any cortex of the area striata type in these adult animals, but he easily recognised it in fœtal and new-born animals. In these it is easy to see that it occupies the same position as in the carnivora, *i.e.* in the gyrus marginalis, between the S. splenialis and S. lateralis, but it crosses the latter.

Finally, in the marsupials the area striata occupies much the same position as in the lower apes, being situated on the dorso-caudal margin of the hemisphere, and more extensive on its lateral

than its median surface. It is also distinguished by its histological structure being very similar to that of the primates, the fourth layer being divided up into three substrata, and by its relatively great extent and its sharp limits.

It has been much more difficult to trace the *area gigantopyramidalis* (the precentral area of Campbell) through the various orders of mammals, as it is often bounded by transition types, and in some species (the lissencephalic apes) two areas overlap. In the higher apes it extends further oralwards, especially into the upper two frontal convolutions, than in man, so that the size of the area relative to the surface area of the frontal lobes is greater than in man. In man it extends to the bottom of the S. Rolandi, but in many of the apes not so deep in the wall of the sulcus, so that in even closely allied species the G. centrales may not be pure homologous fields. Also, in the lemurs, for instance, it extends behind the sulcus which has generally been regarded as the homologue of the S. Rolandi, while in neighbouring species its caudal limit does not reach the sulcus.

As neither the S. cruciatus or S. coronalis of the carnivora have the same topographical relation to the area gigantopyramidalis as the S. Rolandi to this area in man, neither can be regarded as the homologue of the central sulcus. In the majority of the carnivora, in fact, the area gigantopyramidalis lies only behind the S. cruciatus, and in the cat it extends into both the G. sigmoidei anterior and posterior. The S. coronalis never marks its caudal limit, as Köppen and Löwenstein have maintained.

In the ungulates, in contrast to the carnivora, the greater part of the area gigantopyramidalis lies in front of the S. cruciatus.

In the rodents and insectivora the position of the area resembles that in the lower monkeys, but a well-marked fourth layer, which is absent from the typical precentral cortex, extends into the area occupied by large (? Betz) cells. This also occurs in the marsupials, in which the area is relatively very small.

The following statements selected from the general conclusions which end the paper are of interest. Of the different layers of the cortex the greatest variability is found in iv.; the greatest amount of histological differentiation in iii. and v.; and the greatest variation in breadth in vi. Ascending the mammalian series, iii. increases in size, while vi. on the whole diminishes. The degree of development of any cortical area may not be constant in any stage of evolution; the same area may be more similar in animals of distant orders than in allied species. The area gigantopyramidalis and striata are most highly developed in the primates.

The sulci are of much less value in the topographical localisation in the cortex than they have been assumed to be. They may simplify surface localisation, but do not necessarily establish the

limits or positions of cortical structural fields. In many cases the sulci of different classes which have been regarded as homologues owing to their position or course are not homologues.

The value of the work is increased by clarity of description and illustration. GORDON HOLMES.

THE HELWEG-WESTPHAL TRACT. A. GIANNELLI, *Journ. of*
(49) *Ment. Path.*, Vol. viii., No. 1, p. 1.

THE author has examined this tract in the medulla of an idiot with agenesia of the ocular globes and atrophy of some of the olfactory tracts.

At the level of the pyramidal decussation there was found an area of triangular shape at the periphery of the lateral columns, with complete degeneration of the fibres. Within and behind this was a circular rarefied zone separated from the central grey matter by a thin band of normal fibres. At the beginning of the decussation of the sensory tracts the position of these two zones changed, the triangular area lying external to the external end of the accessory median olive, with the rarefied zone immediately behind it. At the level of the complete decussation of the sensory tracts the triangular degenerated area became circular in shape, and then semi-circular, embracing the lower end of the inferior olive, while the posterior degenerated zone was pressed backward against the ascending root of the fifth nerve.

Helweg's tract, therefore, in its upper part, has the shape of a groove (appearing in horizontal sections as a semi-circle embracing the external part of the bulbar olive). The fibres gradually disappeared opposite the lower two-thirds of the bulbar olive, and probably ended in relation with the cells of its dorsal leaflet. From its topography the author would term this tract the Fasciculus Circumolivarius. J. H. HARVEY PIRIE.

THE CONNECTIVE TISSUE CHARACTER OF THE SEPTA OF
(50) **THE SPINAL CORD AS STUDIED BY A NEW STAIN.**
A. R. ALLEN, *Journ. of Nerv. and Ment. Dis.*, Dec. 1906, p. 771.

BY this stain the writer has demonstrated that the posterior median septum and some of the lateral septa contain connective tissue continuous with the pia, and are not exclusively neuroglial. The steps of the method are as follows:—

1. Harden material as for the Nissl method.
2. Sections are placed in ½% aqueous solution of azure II.,

steamed for two to three minutes, and allowed to cool for fifteen minutes.
3. Wash in distilled water.
4. Absolute alcohol until clouds of blue cease to be given off.
5. Place in 20% phenol or toluol (boiling-point 110°-112°) for about one minute.
6. Place in solution of eosin (Grübler w.g.) in absolute alcohol (alcohol 60, eosin 0·02-0·05), and as soon as the change from dark blue to purple becomes pronounced (from 5-60 seconds) remove at once and
7. Place in 25% carbol-xylol for differentiation. Remove before it turns pink.
8. Pure xylol.
9. Mount in balsam in xylol.

The pia is stained a vivid pink, and the sub-pial neuroglia tissue blue. The nerve cells are well stained also, and there is a differentiation in the walls of the blood-vessels, the intima and media staining blue, the adventitia pink.

With material hardened first in formalin, then in Müller's fluid, the Nissl granules are preserved and the axis-cylinder processes are stained blue, but the differentiation between the pia and the sub-pial neuroglial tissue is lost. J. H. HARVEY PIRIE.

PHYSIOLOGY.

PHYSIOLOGIC EFFECTS FOLLOWING SUCCESSIVE ABLATION
(51) **OF ONE FRONTAL LOBE AND ONE CEREBELLAR HEMISPHERE.** G. MINGAZZINI and O. POLIMANTI, *Journ. of Ment. Path.*, Vol. viii., No. 1, p. 14.

THE authors have performed homolateral and contralateral hemi-fronto-cerebellar extirpation in dogs; cerebellar removal followed by frontal, and vice versa. They find that homolateral extirpation is followed by ataxia and asthenia on both sides of the body, but more marked on the side of the extirpation—very similar, in fact, to bilateral cerebellar removal. Contralateral extirpation produces only one-sided effects—that on which the cerebellum is removed—but the disturbances are far more marked than those following isolated hemi-frontal or hemi-cerebellar extirpation. The conclusion seems to be that each frontal lobe has an indirect action on co-ordinate movements of the limbs on the opposite side, and particularly on the anterior limb. J. H. HARVEY PIRIE.

PATHOLOGY.

EARLY APPEARANCE OF PHENOMENA OF REGENERATION
(52) **OF NERVES AFTER THEIR SECTION. (Précocité des Phénomènes de Régénérescence des Nerfs après leur section.)** MARINESCO et MINEA, *Comptes rendus des séances de la Soc. de Biol.*, T. lxi., p. 383.

THIS is a short note dealing with the phenomena of regeneration found twenty-four hours after section of a nerve. Some of the axis-cylinders swell and dissociate longitudinally into fascicles of neurofibrillæ; these interlace with each other and with those of neighbouring axis-cylinders, and may in their turn divide, giving rise to secondary branches, some of which end in a cone of growth.

Another equally early mode of multiplication is by collateral division—fine fibres become detached from the axis-cylinders, constituting the first stage of the spiral apparatus previously described by the authors and by Cajal.

Multiplication by formation of terminal arborisations also occurs at this early stage. In all modes a close relationship is found between the young axones and the "apotrophic" cells.

J. H. HARVEY PIRIE.

CLINICAL NEUROLOGY.

ON SOME OF THE NERVOUS COMPLICATIONS OF THE
(53) **SPECIFIC FEVERS.** Sir THOMAS BARLOW, *Brain*, 1906, p. 303.

SIR THOMAS BARLOW was singularly happy in his choice of a subject for his presidential address to the Neurological Society of the United Kingdom. In the consideration of this wide and fascinating subject the president brings out in striking manner the importance of the recognition of these complications to the general physician as well as to the neurologist. Many of the cases which he uses as illustrating some of the complications of the specific fevers are taken from his own experience, and similar cases are cited from the literature. By correlating the clinical side with the morbid histology, he not only throws direct light on many points, but makes valuable suggestions for the future investigation of many problems as yet unsolved. It is impossible to single out special portions for notice, as the whole address is devoted to a broad and general survey of a field of medicine which as yet has not received the attention it demands and merits.

T. GRAINGER STEWART.

ALCOHOL AND DISEASES OF THE NERVOUS SYSTEM.

(54) **(L'Alcool e le malattie del sistema nervosa.)** L. BIANCHI, Il Tommasi, *Giornale di Biologia e Medicina*, Anno 1, N. 23, 1906.

THIS is a lecture introductory to the author's clinical course upon nervous and mental diseases. The known facts regarding the toxic action of alcohol, the changes the substance undergoes within the body, and its action upon the mental functions, and statistical evidence from various countries bearing upon the relation of alcoholism to disease and crime are first summarised. The author himself assigns to alcoholism in the parents a very potent influence in the causation of idiocy, imbecility, and other less pronounced forms of intellectual defect, as well as of epilepsy, severe hysteria, criminality, etc., in the descendants. In this connection he mentions that he has observed two families, all the members of which suffered from Friedreich's ataxia (which he looks upon as dependent upon imperfect development of the spinal cord, a variety of spinal or spino-cerebellar idiocy corresponding to defective development of the frontal lobes, or the speech areas, in some microcephalic idiots), and the sole cause to which the morbid condition could be attributed was great abuse of wine by the parents. Although alcoholism is less prevalent in the south of Italy than in the north, its percentage incidence in the latter is nevertheless very high, and even alarming. Out of 1814 patients admitted to the asylum at Naples from 1901 to 1905, there were 314 whom Bianchi definitely ascertained to be alcoholics. The author also touches upon the probable causes of the special tendency to the abuse of alcoholic drinks manifested by industrial workers. He thinks that there is perhaps an unconscious effort to make up for deficiency of food by taking alcoholic beverages, or to counteract the mental depression that is produced by the existing intellectual, physical, and social conditions. In the south of Italy, where there are practically no industries, the distribution of alcoholism coincides closely with the malarial zones, and this fact is probably dependent upon the mental and bodily depression induced by malarial infection and deficiency of food. Although acute alcoholic intoxication is very rare in southern Italy, drunkenness from wine is extremely common, and Bianchi considers it is probable that much, not only of the epilepsy, crime, and idiocy that occur in this region, but also of the corpulence, indolence, indifference, rarity of noble impulse, excitability, irascibility and impulsiveness displayed by the population may be due to excessive use of wine. With regard to remedial measures, Bianchi expects more from education than from legislation, and in this process of education of the people he maintains that the doctor must take a leading part. W. FORD ROBERTSON.

A CLINICAL CONTRIBUTION TO OUR KNOWLEDGE OF
(55) **MYASTHENIA GRAVIS. (Contributo clinico alla conoscenza del morbo di Erb-Goldflam.)** E. LEVI, *Rivist. di patol. nerv. e ment.*, 1906, pp. 404 and 441.

THIS is an elaborate and detailed account of nine cases of myasthenia gravis observed by the writer. He analyses the symptomatology of each case with minute care, but no new facts of interest are brought out. He considers the characteristic features of the disease to consist in the intermixture of bulbar symptoms with an external ophthalmoplegia, generally incomplete, and with a peculiar weakness and easy exhaustion of the musculature of the trunk and limbs, usually slow in onset but developing step by step, muscular asthenia always overshadowing paralytic phenomena. Certain muscular groups are attacked by preference; the disease is a remittent one, the myasthenic reaction is frequently present, and at the acme of the disease there may even be a degree of muscular atrophy.

Most cases begin with ocular symptoms, in the form of unilateral or bilateral ptosis or diplopia. And whatever symptom be the initial one of the disease, that one generally remains the most persistent phenomenon throughout the course of the malady.

The author is somewhat confused in his description of the myasthenic facies. He mis-quotes Gowers' description of the "myasthenic smile," and is of opinion that it resembles that of facial myopathy, whereas Gowers clearly points out the difference between the facies in the two diseases. Levi emphasises the occasional presence of slight muscular atrophy in severe cases of myasthenia, so that this feature cannot be regarded as an absolutely differential point in the diagnosis from progressive muscular atrophy. The myasthenic reaction to faradism is by no means a constant phenomenon; it may be absent throughout the entire course of the disease, though this is unusual. The prognosis of the disease, according to Levi, is not so immediately grave as some authors would have us believe. The malady may remain quiescent for sixteen or more years before proceeding to a fatal termination, though this duration is the exception. As to treatment, the main object is the avoidance of fatigue, physical or mental; if difficulty of deglutition appears, special feeding with liquids or semi-solids must be resorted to. Feeding with the stomach-tube is not recommended, because of the liability to produce inhalation pneumonia. Pregnancy is always to be feared, and may be the direct cause of death. Oppenheim has called attention to the frequency of congenital malformations in myasthenic patients; thus, for example, bifid uvula, unilateral congenital ptosis, polydactyly,

webbing of the toes, micrognathy, etc., have all been described. The various pathological appearances in the muscles and lymphoid tissues are referred to, and the writer is of opinion that, notwithstanding the absence of positive pathological appearances in the central nervous system, it is incredible that there is not some obscure change in the nerve elements themselves. A copious bibliography completes a somewhat diffuse paper.

PURVES STEWART.

MYATONIA CONGENITA. [**Zur Kenntnis der sogen. angeborenen** (56) **Muskelschlaffheit, Muskelschwäche (Myohypotonia, Myatonia congenita**).] BERNHARDT, *Neurol. Centralbl.*, Jan. 2, 1907, p. 2.

THE condition under consideration is a very obscure one and the literature is scanty. It is not always quite easy to understand the exact point of Bernhardt's somewhat lengthy argument, but as far as the abstractor can follow him, his main contention is that myatonia is due to degenerative or neuritic changes in the peripheral nerves, as opposed to Oppenheim's view that the disease is a delay in the development of the muscles. Bernhardt recalls a paper written by him in 1904, in which he pointed out that in certain lesions of the nervous system, groups of muscles which were not, and never had been, paralysed, might give the reaction of degeneration. He at the same time described two cases of children in which this state of matter existed; in one case the legs only, in the other the limbs and neck muscles, were paralysed, while all the muscles, including those which could be moved, gave the reaction of degeneration. These cases (which ultimately recovered) he regarded as polyneuritic, and from the peculiarity of the electrical reactions thought that "it [*i.e.* the degenerative or inflammatory process in the peripheral nerves] would probably be one of those forms which, in addition to showing the changes in the peripheral nerves that lead to Wallerian degeneration, also show that form of neuritis described by Gombault as the pre-Wallerian, in which there is a lesion only of the medullary sheaths." In myatonia it seems more likely that there is delayed development of the peripheral nerves than delayed development of the muscles. Bernhardt next refers to Oppenheim's view that there is a congenital defect of the muscles, and then mentions the findings at the autopsy on Spiller's patient. In that case lesion of the thymus was present, and Bernhardt suggests that there may be some connection between this and the disease in question, taking as an analogy the delayed involution of the thymus sometimes met with in myasthenia gravis. The writer next discusses the question of polyneuritis in early infancy, holding that it is not so rare as has been

thought, and hinting (for example) that some of the reported instances of poliomyelitis acuta followed by complete, or almost complete recovery, were really polyneuritic in their nature. In most of the reported cases of myatonia, he states, the electrical reactions are diminished or absent. It is a strong point in favour of the disease being due to nerve lesion that, as in Gombault's "periaxile neuritis," non-paralysed muscles exhibit the same reactions as the paralysed ones do. The following additional points may be noted: In Bernhardt's cases the lower limbs were most affected. The disease occurs only in early life, but whether it is ever actually congenital is doubtful. J. S. FOWLER.

SOME OBSERVATIONS ON PARALYSIS OF THE BRACHIAL PLEXUS. (57) W. B. WARRINGTON and ROBERT JONES, *Lancet*, Dec. 16, p. 1644.

THIS paper is based on the study of 30 cases, their treatment resulting in 11 recoveries—18 were cases of injury (falls on shoulder, etc.), 1 case, which was a dislocation of the shoulder, ending in recovery; 8 were obstetrical in origin, and 4 resulted from neuritis.

In regard to the *pathology* of the lesion, the authors assert that their cases lend support to Clark, Prout, and Taylor in their contention that the laceration, tearing or tension of the nerve tissues is commoner than simple pressure by bone or inflammatory material. Some of the *signs* of loss of nerve function exhibited by these cases shed light on the anatomy and physiology of the brachial plexus; they also have an important bearing on the prognosis of these palsies.

Two cases are mentioned illustrating the absence of sensory defects in lesions of the upper root of the plexus. In one case paralysis of the muscles supplied by the fifth root, without any anæsthesia, resulted from the division of a nerve root during the removal of a large tumour from the neck.

In the other case, exhibiting paralysis, but no sensory defect, during an operation for secondary suture the nerve trunk was divided at the junction of the fifth and sixth roots for the purpose of anastomosis with seventh root: as a result there was no further palsy, but an area of anæsthesia appeared over the deltoid area and over the outer half of hand and lower forearm.

The authors think that Harris's suggestion, in regard to the path taken by the motor fibres of the circumflex and musculocutaneous nerves via the fifth root, and by the sensory fibres of these nerves via the sixth root, is borne out by these two cases.

These two cases also support Harris's view of the extent of the motor supply of the fifth root (prefixed type) embracing, in addi-

tion to the deltoid and forearm flexors, the radial extensors and the pronator radii teres. The assistance afforded by the nature and extent of the *anæsthesia* in the diagnosis of the site of lesion is illustrated by a case in which the muscular palsy and the pupil changes indicated the 8th C. and 1st D. roots as the site of the lesion: the sensory changes, being of the dissociation type, confirmed the *radicular* origin of the lesion. A case where the anæsthesia was similar in distribution, but not of the dissociation type, was mentioned by way of contrast; here a lesion of the ulnar and int.-cutaneous nerves was diagnosed and confirmed.

The authors have studied the behaviour of the paralysed muscles under *electrical stimulation*, and have been impressed by the complexity of the question and the frequent difficulty of giving a correct prognosis.

Two of their cases, in which the affected muscles gave the complete R.D. (Head and Sherren type), showed no sign of recovery in several years; on the other hand, a case exhibiting incomplete R.D. only gives no indication of recovery after seven months.

Having mentioned the important bearing of the site of lesion upon prognosis, the authors enumerate some anatomical facts of interest. For convenience of description the spinal roots are divided into intra-, inter-, and extra-vertebral parts.

The following clinical guides to lesions of these parts are mentioned: (1) pupillary changes result from inter-vertebral lesions; (2) palsy of rhomboids, serratus magnus, and levator anguli scapulae denote a lesion proximal to extra-vertebral portion; (3) paralysis of spinati denotes a lesion proximal to the junction of fifth and sixth roots: this is a more constant and reliable guide than 2.

The authors state that the gravity of the prognosis increases with proximity of the lesion to spinal cord, and diminishes with accessibility of the lesion to surgical interference. The situation of the lesion is stated to be most commonly radicular.

In regard to *treatment*, the importance of mechanical appliances in combination with massage, prior to operation, is insisted upon.

The signs due to loss of nerve function give no indication of the nature of the lesion, whether due to simple pressure upon, or to severe laceration of, nerve tissue. In the latter event, operation would be imperative; whereas in the former case, spontaneous recovery may ensue. Since signs of spontaneous recovery will be manifest within seven months, the authors think that mechanical treatment should be persevered in for at least this length of time, and the withholding of such measures is, in their opinion, reprehensible.

The appliances utilised are a light malleable metal splint for the wrist and a sling: by means of the splint, the wrist can be maintained in the hyper-extended position and the extensor muscles relaxed, and by means of the sling the elbow is kept acutely flexed and the flexors of the forearm relaxed.

This postural shortening of the paralysed muscles in combination with energetic massage places them under the best conditions for recovery; the bandages fixing the splint should overlie tendons only and never the muscle belly.

These methods failing after at least seven months' application, surgical interference should be undertaken if the signs denote accessibility of the lesion.

The authors discuss the various methods of operating for secondary nerve suture—end-to-end junction, anastomosis, nerve transplantation, etc.; the former method they regard as ideal, but not always available in brachial plexus lesions. They draw attention to the fact that part only of the nerve trunk may be involved, and that careful electrical testing of bundles may avoid unnecessary division of nerve fibres: rigid asepsis on the part of assistants applying these tests is insisted upon.

When adhesions are feared, after extensive dissection in fibrous tissues, the nerve ends may be surrounded by sterile foil.

After healing of the operation wound, the postural shortening of lengthened muscles, with massage, should be practised.

Several cases are related where this line of treatment met with success.

The authors arrive at the following conclusions as to prognosis unmodified by surgical treatment:—

1. "That the condition is a serious one, only from 30 to 40 per cent. terminating in useful recovery. That birth palsy of any severity less commonly results in good recovery than the corresponding condition in adults. That the cases due to a local neuritis are a good deal more favourable than when injury is the cause."

2. "That the lesion is commonly severe (laceration, etc.) rather than due to simple pressure."

3. "That a complete block of conduction with a corresponding graver prognosis may be diagnosed from (*a*) a consideration of the extent and kind of anæsthesia (Head and Sherren); (*b*) a study of the electrical reactions, especially with the constant current, but that the presence of full R.D. is compatible with complete recovery; and (*c*) attention to the extent and distribution of muscular palsy.

"The order of gravity of prognosis is lesions of the spinal cord, roots, plexus proper, peripheral nerves."

4. "That if spontaneous recovery happens, some signs of it are visible within seven months in adult palsy, but that in birth palsy

signs of recovery may be delayed for a longer period but are complete within a year. In birth palsies partial recovery is frequent, leaving the residual paralysis to be treated surgically."

AUTHOR'S ABSTRACT.

DISTURBANCE OF SENSATION IN PERIPHERAL FACIAL PALSIES. (58) **(Die Sensibilitätsstörungen bei peripheren Gesichtslähmungen.)** DONATH, *Neurolog. Centralbl.*, Nov. 16, 1906, p. 1039.

BOTH subjective and objective alterations in sensation over the face affected by peripheral palsy have often been noted. In 58 cases of facial paralysis, Scheiber noted 26 in which there was hypæsthesia to touch, pain, and temperature, which concerned the whole of the affected area, more rarely a portion of it, and still more rarely the mucous membrane of the tongue and mouth. Many other, rather differing, results might be cited. Donath has for some years carefully examined all the cases of this condition coming under his observation, paying attention, not merely to the face, but to the whole of the body, with the following result: in 43 facial palsies, facial sensibility was diminished 20 times, unaltered 25 times, increased twice; over the whole of the corresponding half of the body it was diminished in 15 cases, unaltered in 26, and increased in 2. Thus, only in 5 cases (11·6 per cent.) was the diminution in sensation confined to the affected face alone. In 175 cases of peripheral facial palsy which he observed, 75 (42·9 per cent.) suffered from subjective pains over the affected area or over neighbouring areas. They varied in duration from some hours merely to as long as seven months.

It is customary to attribute these subjective and objective changes to the fact of the various anastomoses between facial and sensory nerves, such as the rami communicantes with the auditory, the vidian nerve, the great superficial petrosal, the twig to the tympanic plexus, which runs in the connection between the otic ganglion and the glossopharyngeal, the chorda tympani, the posterior auricular branch which joins the great auricular from the cervical plexus, etc. In 4 out of 10 cases, MacCallan found a direct connection between the glossopharyngeal and facial nerves at the stylomastoid foramen.

Yet this anatomical explanation is unsatisfactory. These anastomoses are more or less constant, whereas the sensory changes in facial palsy are not. When they occur, they ought to be much more pronounced than they often are. Further, occipital pain cannot be thus explained.

The author is of opinion that, in many cases, the pathological

process that has produced the facial palsy simultaneously affects the terminations of the trigeminal and of the cervical plexus and other sensory nerves. S. A. K. WILSON.

THE SO-CALLED ACUTE MULTIPLE SCLEROSIS. [Die sogenannte acute multiple Sclerosis (Encephalo-myelitis periaxialis scleroticans.)] MARBURG (OTTO), Leipzig und Wien, 1906.

(59)

A NUMBER of cases of disseminated organic disease of the nervous system have been recorded which, from their resemblance to disseminated sclerosis, both clinically and pathologically, may be described as acute or subacute multiple sclerosis.

In a monograph of 100 pages, Marburg gives a review of the cases recorded in literature, and adds the reports of three which have come under his own observation.

The disease occurs chiefly between the ages of 20 and 30. The symptoms indicate that there are multiple lesions in the brain and spinal cord; the onset is gradual; there is no fever; and though the affection is progressive, there is a tendency to remission and fluctuations in the symptoms. In the majority of the cases recorded, death occurred within three months—in some cases earlier, in some later.

The author considers that this so-called acute multiple sclerosis is a form of true multiple sclerosis which is characterised by a more rapid course of the disease. The symptoms indicate that it is a form of multiple sclerosis, especially the mode of onset, the advance of the disease in stages, the remissions and intermissions, and the indications of multiple lesions.

The pathological changes are characterised by degeneration of the medullary sheath of the nerve fibres, while the axis-cylinder is relatively intact. At the same time, or soon afterwards, there is proliferation of the neuroglia cells and of cells in the walls of the blood-vessels. The process is analogous to peri-axial neuritis.

The pathological changes are inflammatory, and belong to the group of degenerative inflammatory changes. The nature of the degeneration of the medullary sheath of the nerve fibres indicates that it is due to a "lecitholysis," such as can be produced experimentally by "ferment" action. The pathological changes may be regarded as the result of a toxin. The final stage of the pathological changes is the complete replacement of the degenerated nervous tissues by a finely fibrillated neuroglia tissue, which contains only few nuclei.

The affection is regarded by Marburg as a form of degenerative myelitis, and he suggests for it the name of encephalo-myelitis periaxialis scleroticans.

Three cases are recorded in detail, and eleven excellent illustrations of the histological changes in the nervous system are added.

R. T. WILLIAMSON.

ACUTE DISSEMINATE SCLEROSIS. (Über akut verlaufende (60) multiple Sklerose.) KARL WEGELIN, *Deutsch. Ztschr. für Nervenheilk.*, Bd. xxxi., S. 313.

EDUARD MÜLLER has shewn that the anatomical picture of disseminate sclerosis may be the final stage of quite different processes. For instance, he separates sharply the acute progressive condition following disseminated encephalomyelitis from the true *sclérose en plaques*, on both clinical and anatomical grounds. Wegelin describes a case, however, that shews the great difficulty of making this distinction. The patient was a man of 34. The first symptoms were paræsthesiæ and formication in both legs. Weakness followed, and within six weeks the paraplegia became absolute, both to motility and sensation, from the third rib down. There was at that time no other symptom except slight nystagmus. This state continued for three months; some of the sensory symptoms improved, but marked atrophy of the small hand muscles developed, with reaction of degeneration. Lumbar puncture showed on one occasion 52 cells to the c.cm., on another, 82; 86 per cent. of the latter were lymphocytes. The patient, with broncho-pneumonia, died six months after the first symptoms. In the course of the latter, speech disturbances, paralysis of the right internal rectus and left facial and left hypoglossal made their appearance, though only for two days, as they disappeared before death. At the autopsy, very typical patches of sclerosis were found disseminated through the brain and spinal cord, especially the latter. There was a large area from the first to third dorsal segment, becoming quite complete in the latter situation. The posterior columns, particularly the median, were especially affected. Vessel changes were slight and inconstant, differing thus from those described in most cases of acute multiple sclerosis. The writer considers that they were secondary. A detailed description of the sclerotic foci is given; they differed in no single respect from the classical descriptions of the patches in disseminate sclerosis. The writer controverts Müller's views as to the non-existence of true acute disseminate sclerosis, in detail, and regards his case as conclusive evidence of the existence of such cases. He considers that Müller's grouping of all such cases under the title "secondary sclerosis" is arbitrary, as no distinction can be drawn between the acute and chronic cases.

ERNEST JONES.

CASES OF MENINGITIS WITH ISOLATION OF DIPHTHERIA
(61) **BACILLUS FROM CEREBRO-SPINAL FLUID.** MORRELL and WOLF, *Journ. Amer. Med. Assoc.*, Dec. 29, 1906, p. 2138.

A CHILD, aged 4½ months, was admitted to hospital for meningitis. Forty-five c.c. of turbid cerebro-spinal fluid were removed under high pressure by lumbar puncture. The cellular elements were almost exclusively small mononuclear leucocytes. An organism was isolated showing all the characters of the diphtheria bacillus, including pathogenicity to guinea-pigs. Cultures from the nose and throat, which showed no evidence of inflammation, yielded staphylococci, streptococci, pneumococci, and bacilli resembling those isolated from the cerebro-spinal fluid. The autopsy revealed general miliary tuberculosis. Cultures from the heart blood, peritoneal and pericardial fluids were sterile, but those from the meningeal fluid and exudate at the base of the brain yielded the same organism as that isolated from the cerebro-spinal fluid by lumbar puncture.

The writers think that diphtheria bacilli occur more frequently in the central nervous system than is suspected, and that on careful search they could be found in a large number of cases.

J. D. ROLLESTON.

A DEFINITE CLINICAL VARIETY OF CEREBRAL ARTERIO-
(62) **SCLEROSIS.** JOSEPH COLLINS, *Journ. of Nerv. and Ment. Dis.*, Dec. 1906.

DR COLLINS describes a clinical picture of cases of cerebral arterio-sclerosis, where the cerebral arterio-sclerosis is the primary condition, and in which no changes can be found in the skeletal vessels during life. He states that it forms a definite clinical picture upon which a diagnosis of the condition can be made. The patient complains of headache, varying in degree and character; he is subject to attacks of meaningless laughter, more rarely of crying, without attributable cause. The appearance of the patient is very striking, resembling that of a patient suffering from paralysis agitans, the immobility of the features is very marked. The gait is slow, limited, and shuffling. Mental symptoms come on later, marked by depression and apathy. The reflexes are active, Babinski's sign is sometimes present. The arterial pressure is not necessarily high, and there is sometimes complete absence of visceral and skeletal arterio-sclerosis.

Dr Collins describes two examples of the condition and believes it to be a not uncommon one.

In the autopsies of these cases the chief change is found in the arteries of the brain.

The brain shows shrinking as a rule, in other cases this is not present.

The most striking changes are to be found in the middle cerebral arteries. DUNCAN LORIMER.

DESTRUCTION OF A SUPERIOR CEREBELLAR PEDUNCLE BY A HÆMORRHAGE. (Destruction d'un pédoncle cérébelleux, etc.) (63) A. POROT, *Rev. Neurolog.*, Dec. 15, 1906.

A MAN aged 50, with an alcoholic history, had a sudden and severe attack of vertigo, without loss of consciousness. Vomiting, diplopia, inequality of the pupils (left greater than right), and slowing of the pulse were other early but transient symptoms. At the end of a week, a careful examination was made. The vertigo had lessened, but still reappeared on the slightest movement. The left knee-jerk, exaggerated at first, was now normal, whilst the right was still active. Hearing was a little defective on the right side. In walking, the legs were kept wide apart, and there was a tendency to fall to the right. There were also present on the right side hemiataxia, hemiasynergia, and diadocokinesia; the actual power of the muscles of the right side was practically unaffected.

The patient died of tuberculosis, and at the autopsy the nervous symptoms were found to be due to a hæmorrhage destroying the right superior cerebellar peduncle at its origin from the cerebellum. STANLEY BARNES.

ARTERIO-SCLEROSIS: A SEQUEL TO PSYCHICAL AND PHYSICAL TRAUMA. (Die Arteriosklerose eine Folge des psychischen und physischen Traumas.) (64) O. WATERMANN and FRANZ LUDWIG BAUM, *Neurolog. Centralbl.*, Dec. 16, 1906, Jahrg. xxv., S. 1137.

IT has long been known that arterial changes, with accompanying high blood pressure, are frequently to be met with in neurasthenic and depressive states. The authors maintain, as a result of three years of observation, that the primary change in the vascular system is the raised blood pressure; especially is this so in the case of the traumatic neuroses. This raised blood pressure leads to diminished extensibility and elasticity in the small arteries, with the ultimate production of arterio-sclerosis and its effects. They quote many authorities in support of this view of the

mechanism of arterio-sclerosis. Their own opinion is that traumatic neuroses invariably produce this effect. This is based on the estimation of blood pressure in a large number of cases. Eight of these cases are shortly described. None had evidences of kidney or heart disease. None had been examined before the initial trauma. All had an average blood pressure, estimated on about twenty occasions, each by means of Gärtner's tonometer, about twenty millimetres above the usual.

ERNEST JONES.

CLINICAL CONTRIBUTION TO OUR KNOWLEDGE OF CEREBRAL TUMOURS. (Klinischer Beitrag zur Kenntnis der Hirntumoren.) G. MINGAZZINI, *Monatsschr. f. Psychiat. u. Neurol.*, Bd. xix., H. 5, S. 442.

(65)

CASE 1. *Tumour of the anterior part of the corpus callosum.*—The patient, a man aged 52, a year and a half before coming under observation had a severe attack of vertigo, which recurred twice some months later. After the first attack, weakness in the arms (not in the legs) was noted, which varied in degree, but never entirely disappeared. Later, head pain, frontal and temporal, was complained of: vomiting and convulsions never occurred. When he came under observation, about three months before his death, there were no mental signs except some defect of memory. In hospital, the head pains were violent and constant, the motor disturbances steadily increased, and mental changes—marked apathy, general slowing of mentalisation and defective memory, without any hallucinations or excitement—appeared fairly rapidly, only a few weeks before death. There was marked double optic neuritis; the left side of the face, upper and lower, was paretic; otherwise the cranial nerves were intact. There was no articulatory defect. The muscles of the neck were generally rigid and the head retracted. All limbs were weak and spastic, left much more so than right, and legs more than arms. Practically no ataxia of limbs; no sensory or trophic changes. Romberg's sign was absent; he could not stand upright at all, and, when he tried, he tended to fall back, and to the right. The left knee-jerk was active; the right could not be elicited; there was no ankle- or knee-clonus; the triceps tendon reflexes were fairly active. Lumbar puncture showed enormous increase of cerebro-spinal pressure. The skull was trephined: no relief of symptoms. The autopsy disclosed a gliomatous tumour involving and evidently starting from the anterior half of the corpus callosum, and extending into the brain on either side—further on the right side than on the left—so as to implicate the upper part of the caudate nucleus, the internal capsule, and the lenticular nucleus.

Remarks.—This case fully supports the view of Duret that bilateral hemiparesis, without implication of cranial nerves or sensory disturbance, is characteristic of tumour of the corpus callosum. On the other hand, the signs enumerated by Bristowe, and later by Ransom, as diagnostic of tumours in this region, receive but little confirmation. Thus, contrary to what they state, the general symptoms of cerebral tumour (*e.g.* headache, optic neuritis, vertigo) were very marked; the paresis appeared not in the form of a hemiplegia, but in that of a paraparesis, affecting first the arms and much later the legs; the mental condition of stupor developed not slowly and gradually, but rapidly, and there were never hallucinations or conditions of excitement; there was never any sign of twitchings or convulsions. Schuster gives, as the signs suggestive of a tumour of the anterior part of the corpus callosum, (1) development of mental disturbances long before any motor signs; (2) an isolated and early implication of one or both lower facial branches; (3) rotation of the head to the paralysed side, and general contracture of the neck muscles; (4) greater paralysis of the arms than of the legs; and (5) a gait like cerebellar ataxia. Of these signs, Mingazzini's case exhibited only contracture of the neck muscles and affection of the arms before the legs: possibly, the tendency to retropulsion should also be included, as it occurs in certain cerebellar lesions.

Mingazzini, therefore, concludes that the symptomatology of callosal tumours is still so doubtful that one cannot venture to make a certain diagnosis in any individual case: at present, we can only collect facts. Even the anatomy of the corpus callosum is but little known: thus, it is still an unsolved problem why, when the callosal fibres degenerate in their course within the corpus callosum, the degeneration stops abruptly there, and does not extend to the hemispheres (Mingazzini's observation).

Clinical evidence (obtained from cases of tumour and of congenital absence of the corpus callosum), supported by experimental results, leaves no doubt, Mingazzini thinks, that the motor phenomena seen in cases of tumour of the corpus callosum are due exclusively to involvement of the pyramidal fibres in the internal capsule and lenticular nucleus, not at all to disease of the callosal fibres: the extent to which the centrum ovale is invaded or compressed by the tumour on one or both sides will determine the distribution of the paresis in any case.

Similar evidence leads him to reject the view of Schuster that mental disturbances are a direct symptom of a lesion of the callosal fibres, dementia (sometimes combined with a state of excitement) of lesions of the anterior part, and conditions of delirium in lesions of the posterior part. Schuster explains the absence of mental symptoms in certain recorded cases of agenesia of the corpus

callosum on the ground of functional compensation by other systems: this cannot be accepted, as compensation could be effected only by the anterior commissure, and this has not once been found abnormally developed in such cases (Bianchi).

CASE II. *Tumour in the upper part of the left central convolutions.*—The patient was a man aged 26. Three years previously, slight irritability of temper had appeared—the first sign; soon headache followed, chiefly frontal, and vomiting when the head pain was violent. No other symptom was noted for about two years, when he had attacks of temporary numbness of the right leg ("sensory epilepsy"), followed in a short time by paresis of the same leg, especially of the foot, which gradually diminished but never entirely disappeared. Soon thereafter, clonic spasms of the Jacksonian type occurred, which were at first limited to the right leg, but later spread to the arm and face of the same side. Consciousness was lost in only one attack, which affected mostly the right side of the body and which was followed immediately by complete paralysis of the right leg; this gradually improved until only the muscles of the right foot were paralysed.

The chief points noted, less than a year after the onset of sensory epilepsy, were: No obvious mental symptoms; tenderness of the cranium, especially over the middle and upper parts of the left central convolutions; marked optic neuritis, greater on the left side, and paresis of right lower face—otherwise cranial nerves intact; paresis of right arm, without rigidity; flaccid paresis of whole right leg, with total loss of movements of foot; right knee-jerk much exaggerated, right ankle clonus, absence of Babinski's sign; no gross sensory changes.

Tumour of the upper part of the left central convolutions was diagnosed and found at operation; a glioma, the size of a tangerine, affecting cortex and sub-cortex of this region, adherent to the superior longitudinal sinus and extending to the free margin of the hemisphere, was removed; death occurred six days later from hæmorrhage and consequent diffuse encephalitis.

Attention is drawn to several points. (1) The first symptom noted was slight irritability of temper, which involved the patient in difficulties; Mingazzini thinks that this defect of self-control was a direct result of the incipient disease, and that this consideration is a point of medico-legal interest and importance, not sufficiently recognised. (2) The early appearance of sensory epilepsy, viz. attacks of numbness in the right leg. (3) The occurrence of monoparesis (right leg) before signs of Jacksonian epilepsy; this is not the rule, indeed it is rare in tumours of the central convolutions (Oppenheim); the explanation may be either that the tumour began in the sub-cortex and later spread to the

cortex, or that a sudden hæmorrhage occurred early in the growth of the tumour. (4) The rare occurrence of such a limited crural monoplegia in a lesion of the central convolutions—at one stage, after the severe convulsive attack, paralysis was limited to the right foot; this affords confirmation of the contention of Bruns that paralysis is a more certain localising sign of a lesion in this region than the Jacksonian attacks are.

A. W. MACKINTOSH.

FAMILY PARALYTIC AMAUROTIC IDIOCY AND FAMILY (66) CEREBELLAR ATAXY OF CHILDREN. (Familiäre paralytisch-amaurotische Idiotie und familiäre Kleinhirnataxie des Kindesalters.) H. HIGIER, *Deutsch. Ztschr. für Nervenheilk.*, Bd. xxxi., S. 231.

THE writer recorded nine years ago two cases of Leber's optic atrophy in Jewish brothers. Since then no other sign of nervous affection has developed in them. He now publishes an account of a brother and sister of these former patients. The girl, aged nine, whose first symptom dates to the age of four, had on examination speech defect, cerebellar inco-ordination, tremor, spasticity, Babinski's sign, increased knee-jerks, optic atrophy, nystagmus, and mental deficiency. The boy, aged thirteen months, was perfectly well for the first seven months of his life; the first symptoms were apathy and gradual uselessness of his muscles. At present he rarely moves his arms and never his legs. The muscles are flaccid, but the deep reflexes are much exaggerated. Blindness is present. There is double optic atrophy; at the macula is a white area twice the size of the disc and having in its centre a cherry-red spot. The boy is thus a typical case of the Tay-Sachs disease, while the girl's case should be counted as a heredo-cerebellar ataxy (of Marie). Three hereditary diseases thus occur in four children of the same parents. The writer then embarks on some theoretical speculations, the main thesis of which is the substantial unity of all hereditary diseases of the nervous system.

ERNEST JONES.

A CASE OF GENERAL PARALYSIS WITH FREQUENT EPI- (67) LEPTIFORM CONVULSIONS, WITH OBSERVATIONS ON THE BLOOD PRESSURE. (Ein fall von progressiver Paralyse mit gehäuften epileptiformen Krämpfen nebst Beobachtungen über das Verhalten des Blutdrucks bei solchen Anfällen.) W. PLASKUDA (of Ückermünde), *Allg. Ztschr. f. Psych.*, Bd. 63, H. 2.

THE author measured with the Riva-Rocci apparatus the blood-pressure of a general paralytic immediately before and after

attacks. Just before the attack the pressure rose somewhat, but at the moment when general convulsions began the pressure fell about 10 mm.; during the convulsion the pressure could not be definitely measured, but it apparently rose suddenly with the convulsion and persisted for a few minutes after the rigidity had passed off and then gradually fell. C. MACFIE CAMPBELL.

THE BORDERLAND OF EPILEPSY: MIGRAINE. Sir WILLIAM (68) GOWERS, *British Medical Journal*, Dec. 8, 1906, p. 1617.

THIS is the third lecture on the subject. (See *Rev. of Neurology and Psychiatry*, Vol. iv., pp. 696 and 697.) The writer acknowledges that the term "migraine" has lost its original significance of hemicranial pain and is now applied to all forms of paroxysmal headache. He maintains that migraine and epilepsy are closely related, as is shewn by their alternation, by the occurrence of one condition when the other is relieved, and by the occasional cases in which the same premonition may usher in either one or the other attack. The clinical resemblances of the two conditions and their differential diagnosis are next dealt with. In the worst cases of migraine an ancestral history of gout is rarely absent; in epilepsy the ancestors have suffered from epilepsy or insanity. The *sensory aura of migraine* practically never lasts less than five minutes, and rarely less than fifteen. Its characteristic feature of gradually ascending the arm, leaving numbness behind it, is never seen in epilepsy. It may affect both hands; it never affects the foot, and rarely the lower limb; it never passes down the trunk nor up to the face and head; it is occasionally felt in the lips and even the tongue simultaneously with the arm. In all these points it differs from epilepsy. The *visual aura of migraine* is also longer that that of epilepsy. The dimness of sight is blotchy, irregular, or one-sided, and is prolonged; in epilepsy it is general and momentary. A single flash of light, or multiple momentary bright "stars," so common in epilepsy, are not known in migraine, nor are the elaborate visual sensations of a scene, face, etc. The auras of migraine may occur with no succeeding headache; temporary aphasia may accompany them, as may a vague sense of giddiness. The severe and lasting pain commonly met with in migraine never occurs in epilepsy, nor is the pain of epilepsy one-sided. Vomiting may occur after both conditions, but immediately in the case of epilepsy, and only after some hours in the case of migraine. Other effects of the pain of migraine may be stupor, somnolence, delirium (followed by amnesia), and loss of consciousness.

In the treatment of migraine the writer recommends continued administration of nitroglycerin combined with strychnine.

ERNEST JONES.

POST-DIPHTHERITIC EPILEPSY. DODSON, *Archives of Pediatrics*, (69) Aug. 7, 1906, p. 626.

A GIRL, aged 10, in the second week of diphtheria developed slight spasms, which first appeared in the right arm, and soon became general. They rapidly increased in frequency and severity, until in a few days she was having forty to fifty grand mal seizures in the twenty-four hours. The spasms disappeared within three or four weeks, and the child made a perfect recovery, and never had any trouble of the kind subsequently.

The epileptic seizures are attributed by Dodson to a special selective action of one of the toxines of diphtheria on the cortical cells. J. D. ROLLESTON.

MY CONCEPTION OF HYSTERIA AND HYPNOTISM. (Ma Con-
(70) **ception de l'hystérie et de l'hypnotisme.** BABINSKI, *Arch. gén. de méd.*, Aug. 28, 1906, p. 2187.

HYSTERIA manifests itself in two distinct groups of symptoms, of which the "stigmata" are more or less permanent, the others are transitory. The stigmata, in addition to their fixity, usually develop without the patient's knowledge, and include pharyngeal anæsthesia, hemianarthria, unilateral impairment of the special senses, in particular vision, with concentric restriction of the visual field, monocular diplopia or polyopia, dyschromatopsia for blue and violet, red remaining normal; in addition, one might mention points douloureux, clavus, etc. Fits or attacks, paralyses, contractures, aphonia, mutism, etc., constitute the transitory symptoms, which often appear suddenly under the influence of some emotion, and sometimes disappear with equal suddenness.

Babinski, however, has found in a wide experience that certain modifications of the classical description are necessary, a conclusion frequently arrived at by observers of other generations and of our own. The so-called permanent symptoms may one and all disappear under suitable treatment. They are the result of auto-suggestion in many cases, or rather of the unconscious suggestion of the examining physician, as Bernheim of Nancy has long maintained. The method of examination of the patient is frequently sufficient to evoke in his mind the idea of a hemianæsthesia, or of a visual affection. By carefully studying the best way to conduct an examination, Babinski has found that in not one single new case of hysteria coming under his observation was hemianæsthesia present. Identical results were obtained in regard to restriction of the visual fields. If the patient had not been examined before,

no alteration was forthcoming. It cannot therefore be said that the stigmata of hysteria are either constant or permanent, and therefore any definition based on their presence is likely to be misleading,

Babinski draws an important distinction between suggestion and persuasion. Suggestion signifies the action by which we endeavour to make someone accept or realise an idea which is manifestly illogical. To tell someone whose muscles are functioning normally that he is hemiplegic or paraplegic is to suggest: but to declare to a patient with functional paralysis that he will get better as the result of treatment is to persuade.

The definition which the author proposes is couched in the following terms:—

> Hysteria is a particular psychical state which is capable of giving rise to certain conditions which have features of their own.
>
> It manifests itself in primary and in secondary symptoms.
>
> The former can be reproduced exactly by suggestion in certain subjects, and can be made to disappear under the sole influence of persuasion.
>
> The secondary symptoms are in direct relation to the primary ones.

Conformably to this definition, Babinski excludes from the category of hysteria any symptom which cannot be reproduced by suggestion. It is impossible to evoke nervous attacks associated with lividity of the lips and followed by cutaneous ecchymoses: these cannot therefore be hysterical. The exaggeration of deep reflexes and the occurrence of "spinal epilepsy" cannot be produced by suggestion: these cannot therefore be hysterical. For the same reason he thinks that hysteria can never give rise to paralysis limited to the domain of a peripheral nerve. And so on. Numerous conditions which have been considered hysterical must be excluded from a disease which has been too comprehensive.

Babinski's conception of hypnotism is that it is a psychical state which makes the subject of it susceptible to suggestion from without. It is manifested by phenomena arising from suggestion, which can be made to vanish by persuasion, and which are identical with hysterical phenomena.

There is therefore no difference between the one condition and the other, except in so far as that auto-suggestion may produce hysterical conditions, whereas the question of auto-hypnotisation is problematical. Resort should be made to hypnotism solely in cases which are rebellious to ordinary methods of treatment.

S. A. K. WILSON.

DEFINITION OF THE NATURE OF HYSTERIA. (La définition et la nature de l'hystérie.) SOLLIER, *Arch. gén. de méd.*, Oct. 9, 1906, p. 2585. (71)

IN this communication Sollier proceeds to examine critically the theory of Babinski on the nature of hysteria. He maintains that the latter's definition of the disease is a *petitio principii*. It is equivalent to the following paralogism: "The troubles which can be reproduced by suggestion in certain subjects are hysterical; now, various symptoms noted in cases of hysteria can be reproduced by suggestion in certain subjects and made to disappear by persuasion, therefore *only* those troubles are hysterical which can be reproduced by suggestion and banished by persuasion." In other words, hysterical symptoms can be reproduced by suggestion and made to disappear by persuasion because they are . . . hysterical. Babinski's definition appears to the author to offer two terms for explanation, only one of which is defined. He proceeds to pass various criticisms on Babinski's theory. The latter cannot regard hemianæsthesia as a fixed stigma, because he has often found it absent (when tested by a strong faradic current) in patients to whom the idea has not been suggested by a badly-conducted examination. Sollier emphasises strongly the existence of most varying degrees of hemianæsthesia which certainly escape notice if tested by strong electricity. Sensitiveness to electricity is often present in cases of hysteria when other forms of stimulus evoke no response.

Further, why should anæsthesia be suggested to the patient by an awkward examination? Why not hyperæsthesia? An association of normal cutaneous æsthesia with anæsthesia of deeper tissues is by no means uncommon. That many of the symptoms of the hysterical subject are unknown to him until demonstrated by the physician appears certain. Of various visceral anomalies it is indeed singular that they always consist in modifications of anæsthesia. An hysterical patient who complains of anorexia and gastric pain has usually a degree of cutaneous anæsthesia over the hypogastrium. If this were the result of suggestion, why should it not be hyperæsthesia?

If a condition does not disappear under the influence of persuasion, we have no right to assume that it cannot be hysterical. Sollier has seen bullæ of pemphigus appear in a few hours in a hysterical paraplegia under the influence of intense emotion, and disappear with improvement in the other symptoms, yet Babinski excludes all such phenomena from the category of hysteria, "because they cannot be reproduced by suggestion."

According to Sollier, hysteria is due to inhibition of the functions of the cerebral cortex: variations in degree and extent of this

inhibition explain the variability of hysterical phenomena. Since cortical centres have a double function, psychical and sensori-motor, hysterical phenomena may be accompanied by sensory, motor, visceral, or psychical disturbance. S. A. K. WILSON.

COLOURED VISION AND COLOUR-BLINDNESS IN HYSTERIA.

(72) **(Ueber Farbensehen und Farbenblindheit bei Hysterie.)** L. BREGMAN, *Neurolog. Centralbl.*, Dec. 16, 1906, Jahrg. XXV., S. 1143.

DYSCHROMATOPSIA is a fairly frequent symptom in hysteria. The earliest indication is a concentric retraction of the visual field, most for green and violet, then for red and orange, least for yellow and blue. This colour affection in worse cases goes on to colour-blindness, usually partial, but occasionally total.

The reverse of this condition, namely, the colouring of the field with a particular hue, is much rarer. The colours that occur are yellow, red, green, blue, in this order of frequency.

A combination of the two conditions must be extremely rare, for Bregman can find no record of any case similar to the one he now publishes. The patient was a soldier, aged 30, who in the Japanese campaign incurred a traumatic hysteria. The symptoms of this are described, and the eye manifestations detailed. All objects seemed to the patient to be tinted yellow. With the wool tests red and green were recognised when saturated, but were called yellow when pale; white was called yellow, and both blue and black were called black. There was concentric retraction of the fields, most for blue, least for yellow. The author calls attention to the fact that the fundamental changes, yellow vision and blindness for blue, relate to complementary colours. He attributes this to an excitation state of the cortical area concerned with yellow, with a corresponding depression in that concerned with the complementary colour.

In a second case, that of a soldier, aged 25, all objects appeared as tinted with red. With the right or worse eye no other colours could be distinguished; with the left eye all colours could be distinguished in spite of their suffusion with red. There was no contraction of the fields.

The author refers to his researches on coloured vision in tabetics, which differs from that of hysterics in the following particular. In tabes the field is tinted with green and violet—when an object is first looked at it appears green, then late violet. The green appears in sharply defined patches, the violet in vague clouds, neither affecting the whole field. They are evanescent phenomena, usually lasting over ten minutes in attacks; while in

hysteria the condition is more permanent. In tabes there is diminished acuity of vision for the two affected colours; in hysteria for the complement of the affected colour, though as this occurred in only one case it is not safe to generalise about it.

ERNEST JONES.

A CASE OF HYSTERICAL FEVER. (Ein Fall von hysterischem (73) Fieber.) GOLDFLAM, *Neurolog. Centralbl.*, Nov. 1, 1906, p. 978.

GOLDFLAM'S case was observed as long ago as 1890, and concerns a girl of sixteen, with symptoms leading to a diagnosis of osteomyelitis of the ulna, and to two operations. No sign of disease of bone or marrow was found, but during the whole course of the illness her temperature fluctuated daily, as in pyæmia, from subnormal to 104° F., or higher. After the operations her case was referred by the surgeon to the reporter, who discovered general analgesia, with the exception of the face, anæsthesia and "areflexia" of the mucous membranes, left-sided ovarian tenderness, exaggerated knee-jerks, and pronounced concentric diminution of the visual field. At a later stage the patient gave unmistakable indications of having tampered with her operation wound and with other parts of her body: in short, she was a case of hysteria mutilans. That the temperature was genuine admits of little doubt, for the continued observation of the physician prevented any manœuvring. Further, the author thinks that profuse sweating, which occurred in her case with each fall of temperature, could not be simulated voluntarily. It should be said that the operation wounds remained clean throughout and healed by first intention.

There are numerous references to the literature.

S. A. K. WILSON.

ADIPOSA DOLOROSA; OR, DERCUM'S DISEASE. LAD. HASKO- (74) VEC (Prague), *Rev. Neurolog.*, Dec. 15, 1906.

UNDER this heading the author gives a clinical description of a fairly typical case of Dercum's disease. The patient was a woman of 60, of a remarkably neurasthenic type, and with an enlarged thyroid, in whom the symptoms had been coming on for about ten years.

The author discusses the diagnosis and causation, suggesting that some "functional lesions" of the thyroid, genital, or other glands may have played some part in causing an auto-intoxication.

STANLEY BARNES.

THE RELATION OF SCHOOL WORK TO THE MENTAL FATIGUE OF CHILDREN. (75) B. SACHS (New York), *Journ. of Nerv. and Ment. Dis.*, Dec. 1906.

IN connection with the effect of school work on the nervous disorders of childhood, the author writes strongly condemning the too hasty conclusions arrived at by the pedantic application of psychologic methods to this matter, and further exploited by writers in popular magazines. He states that, after an experience of twenty-one years, and after studying thousands of cases in private and hospital practice, he has never once seen an instance of serious injury done to a child's mental development by any influence of school as such.

Dr Sachs further points out the immense improvement that has taken place in the methods of teaching in the present day. He recognises the fact that mental fatigue is just as normal and to be expected as physical fatigue, that is, when it is transitory, and promptly recovered from after a period of rest.

In speaking so strongly, Dr Sachs is careful to point out that there are naturally many cases in which the particular child is unfit for the life of the school, but he insists that the school must be arranged for the benefit of normal children, and not for the unfit and mentally deficient.

He then points out the great importance of establishing special schools for those children not quite equal to ordinary school life. Dr Sachs is more inclined to blame the generally restless and hurried spirit of American life as the cause of the functional neuroses of childhood, and he credits the school with effecting a quieting and beneficial effect on children. DUNCAN LORIMER.

APHASIA AND DIASCHISIS. (76) VON MONAKOW (Zurich), *Neurol. Centralbl.*, Nov. 16, 1906, p. 1026.

THIS paper is an important contribution to the study of the question of aphasia. The author admits that the more closely the clinical phenomena presented by patients with aphasia are observed, and the more accurately the anatomical changes are examined, the greater is the difficulty of furnishing a satisfactory explanation of the connection between the site of the lesion and the symptoms of aphasia as observed during life. Even with regard to the coarser localisation of aphasia, there are many and by no means inconsiderable contradictions. The number of negative cases, for instance, is much larger than is usually admitted. Still larger is the number of those cases in which, in spite of the per-

sistence or the increase in size of the lesion in the speech region, the aphasic symptoms disappear: and further, the number of cases is very great in which the aphasic symptoms persist for an unusually long time, although the lesion is beyond the limit of the speech region. Paradoxical cases also, such as sensory aphasia with destruction of Broca's convolution, and motor aphasia with the lesion in the temporal lobes, are by no means exceptional; and again, the anatomical conditions for the causation of special forms of aphasia, such as subcortical or transcortical, are in no degree uniform. Finally, the variety as regards affections of intellect shown in cases in which the site of the lesion and its relation to the surrounding parts are quite similar is very striking.

The author shows that the results of his own special observations indicate that mere interruption of continuity of the fibres is by no means sufficient to explain the varieties of form in aphasia and other allied disturbances. We must lay greater weight than hitherto upon the nature of the disease, the mode of its appearance, and the accompanying circulatory, mechanical, toxic, and other factors. It is not admissible, according to him, to attribute the various special groups of aphasia, as is done by Wernicke's school, simply to the interruption or disappearance of certain bundles of fibres or parts of the cortex. He examines and rejects the theory which explains the rapid recovery of speech, in spite of the persistence of the lesion, by the vicarious assumption of the duties of the special centre by other unaffected areas of the cortex. He points out that it is unreasonable to expect that cortical areas, which already have enough to do in the discharge of their own special functions, should in their more or less impaired condition undertake new and complicated duties, such as those of speech. He insists that in order to obtain a firm physiological basis for the localisation of speech, it is necessary to determine accurately—(1) what symptoms must be produced under *all* circumstances by a purely mechanical destruction of various parts of the speech region; and (2) what lesions can produce the more inconstant and temporary aphasic phenomena.

The author draws attention to a special form of action-at-a-distance (Fernwirkung), to which he applies the term *diaschisis*. He distinguishes this from the effect of circulatory disturbances, irritative disturbances due to the products of the disease, or of mechanical, toxic, and other conditions in the neighbourhood, and he defines it as the temporary suspension of function which arises from the local interruption or disturbance of a tract of fibres which directs or carries out the function of a neighbouring part. Such a suspension of function may arise from the interruption of projection or association fibres in such a way that secondary centres, not in themselves directly involved in the lesion, but

connected through fibres with the part involved, are not stimulated from the centre which has been destroyed. The paralysis or abnormal activity thus produced in associated centres not in themselves directly affected, but which under normal conditions act in association with the primary centre of which they are only relatively independent, constitutes diaschisis. These secondary associated centres may lie at a distance from the primary one. Where the secondary centres are comparatively independent, they will correspondingly soon recover their activity; while those symptoms which are directly produced by the injury to the fibres derived from the affected area alone remain.

As an example of diaschisis may be quoted the disappearance of the spinal reflexes on the paralysed limbs immediately after an attack of hemiplegia. In a similar way may be mentioned the bilateral disappearance of knee-jerks in cerebellar disease, in compression of the medulla, and in complete transverse division of the spinal cord. In none of these conditions is the loss permanent, but in all it is due to an interference with higher centres which are physiologically associated with the parts involved. In like manner aphasic symptoms are in the beginning partly those which are permanent and partly those which are of a more or less temporary character. In small, sharply-defined traumatic foci in the speech area, even if bilateral, the permanent symptoms hardly ever contain a psychical element. They are more limited in character and involve simply the mechanism for the direct production of speech. Thus in lesion of Broca's convolution, they would involve slowing of speech, difficulty in forming words, hesitating speech, and in severer cases complete word-dumbness without agraphia. Lesion of Wernicke's convolution would be accompanied by difficulty of speech, paraphasia, logorrhœa, jargonophasia, perseveration (senseless repetition of words), and, when the lesion is bilateral, cortical word-deafness. In such cases the temporary associated symptoms would be those usually called transcortical, *e.g.* disturbance of the complicated process of "Klang-evocation," the power of understanding the sense of a word, or of arranging words into sentences, loss of knowledge of relation between word and object, loss of the finer components of written speech, partial disturbances of orientation in time and place, and even many disturbances of intellect in so far as they are connected with speech. Such "transcortical" symptoms can only be caused by the elective injuries of diffuse and very complicated associated systems, *i.e.* of paths which are themselves slightly or not at all anatomically injured. These transitory symptoms, in so far as they are not "neighbour-symptoms," or due to allied circulatory disturbances, are usually produced by *diaschisis*. It is clear that the degree of diaschisis will vary in different cases. In some cases diaschisis

may be absent, and in others so many arcs may be injured that their activity cannot be re-established. Symptoms of aphasia then may depend more on the diaschisis in distant regions of the brain than on the local lesion itself. This mode of localisation involves that in each special case of aphasia there must be a definite local lesion; but this is not sufficient of itself to produce the symptoms unless it acts in distant associated parts by diaschisis. Besides the local factor, subordinate and accessory factors, such as the general nutrition of brain, circulation, nature of disease, etc., must be taken into consideration. The extent and the greater or less permanence of the temporary symptoms will depend on various factors, such as general functional conditions and power of recovery of the cerebrum.

In this view the *stabile* forms of so-called cortical aphasia will be regarded as being caused by the destruction of Broca's convolution combined with various diaschisis effects which the brain may not be able to overcome entirely. But if, on the other hand, in spite of great extent of original lesion there is more or less complete recovery of speech, this depends not on vicarious intervention or education of corresponding convolutions of the other side, or any other part of the cortex, but from a retrogression of the original diaschisis. In completely negative cases we must assume absence of all diaschisis in an otherwise normally active brain. In such cases there must no doubt remain slight residual disturbances which can only be discovered by fine methods of investigation.

The conception of diaschisis shows that for the mechanism of speech much more extensive cortical areas are involved than has been hitherto assumed, but that the speech function can be easily affected by injury to certain sharply localised groups of bundles and cortical areas by diaschisis. This is to be explained by preformed, extensive cortical innervation paths, but such local anatomical "nodal" points are not therefore to be regarded as special workshops for speech. Thus a speech centre would simply be a *locus minoris resistentiæ*. In every case of interruption of these paths for speech innervation, there will therefore be a danger of their activity being temporarily affected. With a local lesion and an otherwise sound brain, the greater part of the loss of speech will in a short time be recovered from.

The author hopes that the principle of diaschisis will be of assistance in clearing up our views as to the mode of action of cerebral lesions on the function of speech. ALEXANDER BRUCE.

THE VISION-FIELDS IN CASES OF INDIRECT OR INCOMPLETE (77) LESIONS OF THE OPTIC SYSTEM. COLIN K. RUSSEL (Montreal), *Journ. of Nerv. and Ment. Dis.*, Dec. 1906.

IN an article on the state of the fields of vision in lesions affecting the optic system, the author begins by a short account of the

most recent views on the anatomy of the region. He traces back the macular fibres, and discusses their ultimate distribution. He further points out the comparative unimportance of the pulvinar and anterior corpora quadrigemina in regard to vision, as compared with the external geniculate body. He then discusses the question of the cortical representation of the retina in the occipital lobe, and reviews the opinions on this matter.

A knowledge of their anatomical relations is of extreme importance in regard to cerebral localisation, from the clinical conditions. "The fields and the acuity of vision vary very materially, according as the lesion affects the optic nerve tract or the radiations."

Dr Russel then gives accounts of six cases of lesions in the optic system, with a description of the clinical symptoms, the state of the optic discs, and the alterations in the fields of vision. In two of these lesions (chiasmal), a tumour of the pituitary gland was found, which was operated on by Sir Victor Horsley. The other cases were examples of lesions in different parts of the optic system, and showed correspondingly different clinical manifestations.

DUNCAN LORIMER.

MINERS' NYSTAGMUS. A. CHRISTIE REID, *Brain*, Vol. xxix., (78) 1906.

THE theory of the causation of miners' nystagmus which has received most general support is that of Mr Simeon Snell, which makes it out to be a myopathy induced by chronic fatigue of the muscles, produced by working in a strained position. On this theory, defective illumination is only of subsidiary importance. Such a theory separates miners' nystagmus from certain other forms of the disease which would naturally seem to be closely allied to it, such as the nystagmus which comes as the result of corneal opacities developing in early childhood, and any theory which might lead to the correlation of these different forms will naturally be examined in a spirit of benevolent criticism. An attempt to arrive at some such theory is given by Dr Reid in his interesting paper. He points out, to begin with, that much evidence has been adduced to show that insufficiency of light is the more important factor, and strained position of the eyeballs the less important. His theory is "that the affection is due to a disturbance of the centres having to do with the equilibration of the eyeball." With dark adaptation the superiority of visual acuity in the fovea disappears; in fact, becomes lower than the rest of the retina. This is a factor of prime importance. Dr Reid next points out the close interdependence of equilibration of the eyes with equilibration of the body. In a miner's life both those

factors are met with. There is dim illumination and a constant tendency to disturbances of body equilibrium. Thirdly, there is a more or less rhythmical series of movements performed by body and head with eyes fixed. These, he considers, tend to produce the rhythmicity in character of the nystagmus. The onset is usually precipitated by debilitating influences of any kind.

LESLIE PATON.

MYOPATHY FROM JERKINGS OF THE DELTOID DURING THE (79) MOVEMENTS OF THE SHOULDER. (Myopathie par Sursauts Deltoidiens pendant les mouvements de l'épaule.) GUERMONPREZ, *Gaz. des Hôp.*, Oct. 11, 1906, p. 1383.

A CASE illustrative of the errors likely to occur in the awarding of compensation to workmen for injuries sustained at their employment. A mill-worker suffered from weakness and cracklings of the right shoulder, supposed to be due to a sprain sustained at his work; but on inquiry it was found that no real sprain had occurred, and on examination it was discovered that both shoulders were affected, that the cracklings only occurred when the arms were in a certain position, and that their seat was the deeper part of the deltoid muscle. There was no connection between these muscular jerkings and any sprain or other injury, and it was merely a coincidence that the crackling had been first noticed while the man was engaged at his work.

HENRY J. DUNBAR.

DISORDERS OF SWEAT SECRETION IN DISEASES OF THE (80) SPINAL CORD. (Schweissanomalien bei Rückenmarkskrankheiten.) H. HIGIER, *Neurol. Centralbl.*, Nr. 1, 1907, p. 19.

THE author, who published an article in the *Deutsche Ztschr. f. Nervenheilk.*, Nr. 20, 1901, entitled, "Zur Klinik der Schweissanomalien bei Poliomyelitis anterior (spinale Kinderlähmung) und posterior (Herpes zoster)," is of opinion that anomalies of sweat secretion, such as anidrosis, hyperidrosis, and paradoxical sweat secretion, would be much more frequently discovered and used for diagnostic purposes if they were searched for and duly considered. According to him the central origin of sweat secretion is located on each side of the cortex, and connected with subsidiary centres in the medulla oblongata, where the principal centres for the four extremities are found. The spinal cord contains throughout almost its whole length either centres or fibres for sweating. He records four cases of sweat anomalies. (1) A case of a sudden paralysis of the lower extremities, associated

with an increasing optic neuritis, which produced bilateral amaurosis. On using means to induce sweating, it was found that the lower, paralysed part of the body remained practically perfectly dry, while the remainder of the body perspired profusely. (2) A man of forty-one years of age, in whom a fall from a considerable height was followed by a flaccid motor paralysis of the lower extremities, with loss of sensibility, especially for pain and temperature, with muscular atrophy, weakness of the tendon reflexes, and loss of the lower organic reflexes. The lower half of the body was found to remain perfectly dry, when profuse perspiration was present in the other half. (3) A woman of fifty-two, who had suffered for twenty-five years from progressive weakness in the right hand. She showed a right claw-hand, dissociated disturbances of sensibility in both upper extremities and the right half of the face, and indications of bulbar paralysis. There was loss of sweating on the right arm and the right half of the trunk. Case was probably one of syringomyelia, extending upwards and downwards from the cervical enlargement. (4) A man of forty-eight, from whom a malignant left kidney had been removed five months previously. Suffered for nine months from a flaccid paralysis of the lower extremities, which was first painful and later painless, in which there was loss of all the reflexes, deep and organic, and loss of sensibility in the legs, with bed-sores on the sacrum, absorption from which had produced a severe sweating in the face and the upper part of the body, but left the lower extremities and the lower half of the trunk entirely dry.

The author points out that the secretion of the sweat glands is not to be regarded as a mere physical process of filtration, brought about only by an indirect influence of the nervous system through vaso-dilatation. He offers no opinion as to whether the centres are situated in the anterior horn near the motor ganglion cells, or at the basis of the posterior horns. He shows that the paths for the secretory nerves may be interrupted either in their peripheral course or in the sympathetic chain in the intra-vertebral ganglion, or, finally, in the grey matter of the spinal cord or in the cerebral cortex.

In the paper published by the reviewer and Dr Pirie in the January number of this Review, the opinion is expressed that the spinal centres for the sweating fibres of the lower half of the body are probably situated in the intermedio-lateral cell-system of the lower dorsal and upper lumbar segments of the cord. The writers agreed with Higier in thinking that the loss of sweating depends directly upon the destruction of special centres for the function, and not upon any indirect influence of vaso-motor or vaso-dilator fibres. ALEXANDER BRUCE.

PATHOLOGY OF THE ANKYLOSES AND PARTICULARLY OF
(81) **VERTEBRAL ANKYLOSES. (Pathogénie des Ankyloses et particulièrement des ankyloses vertébrales.)** ANDRÉ LERI, *Congrés de Lyon*, Aug. 2-7, 1906.

THIS monograph concerns itself solely with true osseous ankylosis of the vertebral column, but the general etiology of osseous ankylosis in any part of the body is first summarised, and the importance of ossification as a secondary and compensatory factor in the "functional adaptation" of diseased or injured joints is emphasised. Local vertebral ankylosis is much more common than general, and may be secondary to tubercle, syphilis, chronic rheumatism, gout, traumatism, and lastly to scoliosis, when the ossification occurs in the concavity of the curve. *Hereditary traumatic kyphosis* is an example of general vertebral ankylosis and is probably due to ossification following injury to the anterior vertebral ligaments as a result of sudden straightening of the normal (or exaggerated) kyphotic curves. More common causes of general ankylosis are chronic rheumatism and spondylitis deformans. These the writer considers essentially different from one another. In chronic rheumatism there is great enlargement of every part of the vertebræ, with irregular protuberances and osteophytic outgrowths and ossification of the intervertebral discs. In spondylitis deformans there is an osseous rarefaction, the ossification of the ligaments being compensatory, and there are no osteophytes formed; the bones of the pelvis are softened and the pelvis deformed. The former disease is inflammatory in origin, the first stage being a rheumatic arthritis: the latter consists in an ossification of the ligamentary structures to compensate for the softening of the vertebræ—a "functional adaptation." The consideration of these two conditions is entered into in great detail. The paper is illustrated by photographs and drawings.

HENRY J. DUNBAR.

PSYCHIATRY.

ON COMBINED PSYCHOSES. (Zur Lehre von den kombinierten
(82) **Psychosen.)** E. STRANSKY (of Vienna), *Allg. Ztschr. f. Psych.*, Bd. 63, H. 1.

SINCE psychiatry has ceased to be satisfied with the description and analysis of symptomatological pictures, and has begun to group mental disorders according to the broader lines of their general development, clinical course, and symptomatology, combined psychoses have not been in favour. Cases may present symptom-complexes containing elements belonging to different mental disorders, but that does not justify the diagnosis of two indepen-

dent and separate disease processes. It is true that a definite psychosis, with well-marked features, may develop in a person who has presented congenital anomalies of disposition; also a mental disorder may become complicated by the effect of various influences, alcohol poisoning, arterio-sclerotic degeneration, etc.; these latter are mere accessory additions. The possibility of the combination of an endogenous with an exogenous disease has been emphasised by some authors, but such combinations seem to be rare, *e.g.* the combination of manic-depressive insanity with general paralysis, or the combination of true paranoia with general paralysis. In such cases one has the right to talk of two psychoses. Other combinations are also possible—*e.g.* an amentia in the course of chronic paranoia, and the author gives the record of such a case. Besides the simultaneous combination, there may be the successive combination of two psychoses. The record is given of a patient with bad heredity who, after a first manic attack, recovered for two years, but later had a second typical manic attack, which, however, after a few months began to include extraneous elements; patient now uttered confused delusions of a religious colouring, had hallucinations of various senses, talked in a confused manner, producing more of a word-salad instead of the previous flight of ideas. A typical catatonic picture gradually developed, with alternation of excited and stuporous stages, with cataleptic phenomena, automatic response to commands, negativistic behaviour, dilapidated and drivelling utterances, unmotived assaults. The patient has remained in the same condition for over a year.

C. MACFIE CAMPBELL.

A CASE OF DOUBLE CONSCIOUSNESS (AMNESIC TYPE, WITH FABRICATION OF MEMORY). (83) E. ANGEL (of Rochester), *Journ. of Abnorm. Psych.*, Oct. 1906.

THE case is one of a man 25 years old, who suddenly left his newly-married wife, and after about two weeks was discovered busily engaged canvassing in a town at some distance. The clue to his whereabouts was given by an absolutely false telegram which he himself had sent, dated from a non-existent hospital. When found, patient was in a peculiar condition similar to the hypnotic state. When examined by the author he gave three different accounts of his life. All of his accounts showed "subjective unconscious falsification of memory" for the actual happenings of a somewhat uneventful existence. He filled the gaps with "visions and plausible impossibilities."

For example, in his first account, he referred in a most convincing way to the death of his mother and his twin sister, the suicide of his father, his own training in King's College, and his sub-

sequent enlistment in the Canadian troops in the Boer War. All of these ideas were creations of an unstable imagination.

In his second account he gave an equally false statement of his life, with an imaginative use of actual information with which he was confronted by a near relative; he is living the life of one Robbins, his name is no longer Rawlins, and the history which he first gave belongs not to himself, but to Robbins who is dead. According to his own account, he himself (Rawlins) and Robbins were associated as students in M'Gill University, and as patients in a hospital in Winnipeg. Robbins died and left him (Rawlins) to suffer his sorrows, pay his bills, and inherit his estate. Facts show that Robbins is a myth, and that the patient was never a student at M'Gill University, nor a patient at Winnipeg.

His third account is written after the "full recovery of his normal mentality." He admits that his name is Rawlins and not Robbins, that his mother, brothers, and sisters still live, and that his father did not commit suicide, but died from natural causes. He still insists that he was a student at M'Gill University, but admits that from the time he entered the university up to the present time there have been periods of long duration for which he cannot account. Angel emphasises his belief in the honesty and frankness of the patient in giving these accounts, and declares that in such cases of so-called dual personality, much that has been accepted as actual occurrence during the dispossession of the ego could be proven to be an illusion, if the facts were sufficiently investigated. C. H. HOLMES.

ON THE USE OF ALCOHOL AS AN AID TO DIAGNOSIS IN

(84) **MENTAL DISORDERS. (Über Alkoholversuche bei Beurteilung zweifelhafter Geisteszustände.)** TOMASCHNY (of Treptow), *Allg. Ztschr. f. Psych.*, Bd. 63, H. 5.

THE author recommends the experimental use of alcohol for diagnostic purposes, especially in certain medico-legal cases, where it is important to determine the responsibility of the individual and to ascertain whether he shows a pathological reaction to alcohol which may diminish his responsibility for acts committed. He records some interesting cases. The first patient had stolen a watch, and in prison had by his wild behaviour raised the suspicion of simulation. After 14 days' observation in the hospital, during which time he was irritable, inaccessible, and scolding, his mood suddenly changed, he became quite agreeable, claimed that he had an amnesia for the whole period, including the theft. After 5 weeks of total abstinence he was given a liberal dose of schnapps; about 36 hours later he had three epileptic attacks. The second patient had in a fit of irritation killed his child. During

5 weeks' observation he showed no striking peculiarity in behaviour; he was then given at lunch a bottle of beer and five small glasses of schnapps. When examined half an hour later he showed marked irritability and was extremely threatening. This condition lasted for a few hours. Five hours later he was again normal and denied any memory of having been irritable to the physician. Under the influence of alcohol his pupils became irregular. In another case the patient, who asserted that he was subject to epileptic attacks, and complained of numerous subjective complaints, said that he had had to give up a good position owing to epilepsy. An experimental dose of alcohol caused patient to cheerfully narrate the truth about his past; he now boasted how much alcohol he could stand, while previously he had professed great intolerance towards alcohol. His pupils showed nothing abnormal, The author emphasises the importance of pupillary signs under the influence of alcohol as pointing to a pathological reaction.

C. MACFIE CAMPBELL.

Reviews

NERVOUS DISEASE AND THE PUBLIC. (Nervenkrankheit und Lektüre. Nervenleiden und Erziehung. Die ersten Zeichen der Nervosität des Kindesalters.) Prof. H. OPPENHEIM. Pp. 119. Berlin: S. Krager. Edinburgh: Otto Schulze & Co. 1907.

THE three lectures on "Nervous Disease and Reading," "Nervous Affection and Education," and "The First Signs of the Neuroses in Childhood," contained in Prof. Oppenheim's small book, are expressly prophylactic in aim; and though their author says perhaps too much in affirming that "die Nervosität die Krankheit unserer Zeit ist"—witness the extraordinary hysterical epidemics of the Middle Ages, and the fact that many of the lower races are singularly prone to hysteria—there can be no question that this branch of preventive medicine is one of pressing, and as certainly of increasing, importance. The first lecture treats of the influence of literature, and particularly of the daily press, on nervous subjects, and, whilst admitting the right of the public to a knowledge of scientific discoveries in the department of medicine, he inveighs strongly against the detailed publication in the daily journals of contributions to medical magazines, congresses, and the like, as fostering hypochondriacal fears, or, in the sexual sphere, exciting a prurient imagination, and urges his medical confrères to join in discouraging this tendency. Not only in ephemeral literature, but in modern poetry, the drama, belles-lettres, and particularly in novels, he finds a widespread, unhealthy appetite for the morbid

and subtly indecent; pathological details, which might at one time have been given incidentally with blunt candour, now forming the sole motive of the book or play. In no wise concerned with public morality, but merely in so far as these tendencies find expression in the consulting-room, he finds here just grounds for their condemnation, and gives instances where neurasthenic and hysterical individuals have found here nurture for their complaints. The gist of the lectures is given in a quotation from Schopenhauer, "Um das Gute zu lesen, ist eine Bedingung, dass man das Schlechte nicht lese."

If it be true that medicine begins by being curative and ends in being preventive, it is essential that preventative measures should be instituted early in childhood, and should extend through what Dr Clouston in his "Mental Hygiene" calls the formative period of life. In his second lecture Prof. Oppenheim carefully considers the usual educational methods from the standpoint of the neurologist, and naturally finds considerable room for criticism. In particular the necessity for attention to the affective as well as to the purely intellectual side of development is emphasised—a point customarily disregarded by the pedagogue. Perhaps the most valuable part of this book is the third lecture, in which, out of his wide experience, the author describes the earliest symptoms of the neuroses in childhood (excluding organic brain diseases and the insanities), *e.g.* emotional reactions and their deviations from the normal in form and range, particularly the phobias and their motor expression, the liability to hallucinatory delirium, the tendency to imaginative lying, the so-named *pseudologia phantastica*, the disorders of sleep, the tics, and many habitual motor eccentricities.

Incidentally, Prof. Oppenheim states that he has been struck by the extraordinary liability of the offspring of mixed marriages, particularly between Jews and Christians, to nervous affections. This seems a point of some importance, and it is unfortunate that Prof. Oppenheim is unable to give his opinion statistical support.[1]

R. CUNYNGHAM BROWN.

DÉGÉNÉRESCENCE MENTALE ET HYSTÉRIE. LES EMPOISONNEUSES; ÉTUDE PSYCHOLOGIQUE ET MÉDICO-LÉGALE. Dr RENÉ CHARPENTIER. Pp. 227. Paris: G. Steinheil. Edinburgh: Otto Schulze & Co. 1906.

THROUGHOUT the ages, poison has been a favourite weapon of women, whether the object has been to pursue intrigues, to gratify

[1] Prof. Karl Pearson, in reply to an enquiry from the reviewer, states that he has no data on this point, and suggests that medical men in India might give much information as to Eurasians, whilst in certain districts in London the results of marriages between English and German and English and Jews might be observed.

feelings of revenge or jealousy, or merely in obedience to deep-seated pathological impulses. The women who have become notorious in history for the crime of poisoning have often been found possessed of a peculiar mentality. It is only within the last century that the records of the investigations of such crimes have contained the report of a thorough medical examination of the author of the crime with a view to determine responsibility. The cases on record form a good starting-point for the analysis of the mental constitution and idiosyncrasies of this group of criminals; while a historical review shows many analogous cases, where, however, the mental diagnosis must be retrospective. The author of this study of female poisoners runs over the names famous in history from this point of view, and precedes his historical review with examples from classical mythology. The crimes of ancient Greece and Rome are shortly referred to; in Rome especially there were such epidemics of poisoning that special laws were made to meet the danger. No poisoners are better known, perhaps, than the infamous Borgias, but the author quotes the passage where Gregorovius defends Lucretia Borgia, attributing her crimes to her unfortunate environment. A special chapter is devoted to the notorious Marquise de Brinvilliers, who combined a profound skill in simulation with a moral anæsthesia. On a par with her crimes were those of the hysterical Madame de Montespan.

After finishing the historical account, Charpentier takes up the clinical analysis of this group of hysterical degenerates; he endeavours to separate the hysterical incidents from the stigmata of the degenerate constitution; from the latter point of view these persons show kinship with the moral imbecile. Their hysterical mentality is shown by their suggestibility and their well-marked tendency to mental dissociation. The intellect of the cases discussed showed at the same time evidence of considerable brightness and lapses leading to coarse blunders. Aboulia coexisted with obstinacy, and affective anæsthesia with an excessive susceptibility to impressions.

In the third division of the book, the author discusses the medico-legal questions which such cases raise. These unfortunate people are suited neither for the prison nor for the hospital for the insane; they are abnormal beings, who for their own sake and for the sake of society should be detained in special asylums, and the conditions under which a return to social life might be allowed could be determined by the physician in charge, under the authority of the courts.

The book is written in an agreeable style, contains some interesting illustrations, and presents in language not too technical a subject which is of considerable social and medical interest.

C. MACFIE CAMPBELL.

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Review
of
Neurology and Psychiatry

Original Articles

TABETIC ATROPHY OF THE AUDITORY NERVE.

By Sir WILLIAM R. GOWERS, M.D., F.R.S.

LABYRINTHINE deafness is not rare in those who suffer from tabes, but seldom can be ascribed, with any confidence, to atrophy of the auditory nerve, analogous to that which is so common in the optic nerve. It may be suspected when the deafness is bilateral and progressive, but these features do not justify more than a suspicion. Two characters, however, make the diagnosis probable. These are: (1) a progressive reduction of hearing from above and below, analogous to concentric restriction of the fields of vision; (2) a loss in the centre of the range, analogous to a central scotoma in vision. Both are rare. Their significance is especially great if the deafness accompanies optic nerve atrophy. This indicates that the morbid influence which induces tabes is of a character to affect the nerves of special sense, especially the optic nerve, in greater degree than the sensory nerves of the muscle.

It is a matter of common observation that in tabes, if the optic nerves suffer, the legs often escape, and vice versa. It was said by Benedikt that the relation is one of cause and effect, that the affection of one saves the other from suffering. This has indeed been called "Benedikt's law." I think, as I pointed out many years ago, that the true explanation is a difference in the cause. If we assume that this is a post-syphilitic toxine,

as is probable, the marked difference in the degree of the various symptoms of tabes can only be explained by differences in the character of the toxine. Thus only can we understand the fact that one patient may suffer intensely from pains, another hardly at all; that in one laryngeal and gastric crises may be frequent, and in another never occur. The difference in the toxine may be ultra-minute, and yet adequate to act chiefly on nerve structures that differ in susceptibility in corresponding degree. Thus the inverse relation between the affection of the eyes and of the limbs is not a direct connection, as Benedikt supposed, but an indirect consequence of the nature of their cause. This view is confirmed by the fact that exceptions are not rare. The optic nerves may suffer when there is extreme ataxy.

I. *Bi-terminal Reduction of Hearing*,[1] analogous to concentric restriction[2] of the field of vision. In one case of this kind the patient, when first seen, could hear no note above E^1 (660 vibrations)[3] or below G_1 (198 vibrations). A few months later the range of hearing had become still further reduced, but from below, not from above; hearing was limited by E (330) and E^1 (660), the two E notes within the treble. Even E flat was inaudible. A few months later even this octave had disappeared and he was totally deaf on that side.

II. *Auditory Scotoma.*—A loss in the middle of the range of hearing, analogous to a scotoma in the middle of the field of vision, is also, possibly, evidence of primary atrophy of the nerve. It is rare, and its significance may not be beyond question. The following case is therefore important, because other facts support the assumption of primary atrophy. I will mention very briefly the chief symptoms.

The patient was first seen in 1899, when 57. There was a strong suspicion of syphilis at 19. The right knee-jerk was absent, the left fair. Sensibility to pain was defective on this leg and on the right side of the abdomen; it was almost lost on

[1] "Bi-terminal" seems the only fit adjective for "at both ends."

[2] The word so often employed, "concentric limitation of the field," is undesirable, because a field without limit must be very rare indeed.

[3] This rotation is that which I urged, as most convenient and least liable to error, in this Review for April 1903. In it the unnumbered C is the middle C, *i.e.* that between the treble and the bass (256 vibrations); C^1 and C_1 are respectively in the second space from the top of the treble and from the bottom of the bass staves. The number of vibrations is easily ascertained by remembering that $C_2 = 33$; therefore $C_2 = 66$, $C_1 = 132$, $C = 264$, etc.

the thigh. Tactile sensibility was normal. There was very slight dystasia (Rombergism). Micturition was tardy; sexual power lost. The pupils were small, inactive to light, acting with accommodation. His optic discs were pale, the right more than the left. Vision, corrected for presbyopia, right $\frac{1}{5}$; left quite good. Fields normal. A central scotoma, relative, was found in the right eye, larger for red.

It is needless to follow the case in detail. The tabetic symptoms have scarcely increased. Indeed, the only change is that the left knee-jerk after a time could only be obtained with reinforcement, and is now lost. Vision lessened a little during the first year; the right scotoma became absolute, irregular in form, extending to about 10°, and the field lessened above and outwards. In the left eye, central dimness developed, with a larger loss for red around it. Then his vision became stationary for about three years. During the last three years it has failed steadily, and during the last six months rapidly. The peripheral restriction of the right field steadily increased, and vision became dim in the part between it and the scotoma, so that three months ago he could barely see light with this eye. The scotoma in the left became absolute, but beyond it he could make out $\frac{1}{36}$. Since then, peripheral vision has rapidly passed away in this eye also, and now he can only discern light from darkness. His optic discs have long presented the appearance of advanced atrophy. I have given these details on account of the greater affection of the right side in both legs and eyes, and of the failure of sight by a central loss. I should add that he had always been a very small smoker.

During the last year he has noticed that his hearing was not so good as formerly, especially for the high-pitched voices of children. On testing his hearing it was found to have a range unusually high for his age (now 65). He could hear Galton's whistle (the high whistle-sound being carefully distinguished from the sound of blowing) with each ear up to 12,000 vibrations. With the right ear, on passing downwards, he could hear each sound to 7000 vibrations, but no lower note. He could not hear the C^3 tuning-fork (2112), but could the C^2 (1056) and all lower notes, through the bone as well as through the air. C^3 was not heard even through the bone. On the left side there was a similar loss, but less extensive. He could hear C^2, but not

C^3, but the loss only extended to 5000 vibrations, which he could hear, and higher notes up to 12,000. On this side also the result was the same through the air and the bone.

The association of this auditory loss with the central scotoma by which sight failed, first and chiefly, is remarkable. It constitutes strong evidence that the auditory scotoma was the result of commencing primary atrophy of the nerve. At the same time the subject is a difficult one. In this case the loss was at the region of the highest tuning-fork available, probably about two octaves. This region of the range of hearing is not comparable to the central region of the field of vision occupied by the visual scotoma. There is no special functional capacity at that part of the range of hearing as there is in the field of vision. But failure of central vision in this manner is not common in tabes. More frequently there is a progressive restriction of the field, with general failure. The morbid changes in tabetic atrophy with a central scotoma have not, I think, been ascertained. It may be that they are independent of function, and of a character that the auditory nerve may also undergo. Thus the apparent correspondence in the visual and auditory failure may be more than apparent, and the seeming significance of the auditory scotoma may be real.

ALCOHOLIC PSYCHOSES: A STUDY OF THEIR MECHANISM AND OF THEIR RELATION TO OTHER MENTAL DISORDERS.[1]

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THIS study was prompted by the difficulty encountered in the analysis of atypical psychoses, in which alcohol has played a prominent part. We observe every possible symptom-complex as an apparently more or less direct result of alcoholic excess; in many cases this excess is the first symptom of the psychosis, and not its cause; the exact facts as to the patient's habits are often not obtainable; it thus becomes important to know what conclusions we are justified in drawing from the clinical picture

[1] Read at a Conference of State Hospital Physicians at Manhattan State Hospital, Ward's Island, N.Y.C., December 12, 1906.

itself, as to the part alcohol has played in the etiology. An attempt is made in this study to gain a bird's-eye view of the group as a whole, and to trace through the maze of diverse symptoms and sub-groups some common element to assist us in sizing up doubtful cases.

Speaking of acute alcoholic intoxication, Kraepelin says: "The effect of alcoholic poisoning upon the mind, in so far as it is known, consists essentially in the impairment of the grasp, retention, and elaboration of external impressions, and in facilitating the release of the impulses of the will . . . as all good observers agree, even very small quantities of alcohol materially impair the efficiency for high-grade mental effort. A person is less able to collect his thoughts and to follow an involved sequence of ideas. With further poisoning the impairment of grasp and reason becomes still more pronounced. The individual no longer rightly understands what is said to him and what goes on about him, cannot apply his attention nor follow a train of thought . . . as the loss of grasp and reason progresses, he becomes insensitive, irrational, and finally completely unconscious. . . . The psychomotor irritative symptoms keep pace with the intellectual disturbance. They begin with a mild exhilaration, which is felt after the ingestion of even small doses of alcohol, with the loss of the delicate inhibition which regulates with accuracy his daily conduct and manner. He becomes carefree, is in good spirits, cheerful, unrestrained in speech and action, courageous, even reckless. On account of the facility of the release of motor impulses, his strength and capacity for work seem to be increased in contrast with their actual material diminution. From this arises the unfortunate, widely diffused, but entirely erroneous impression that alcohol is a stimulant. . . .

"In the affective field the first stage of intoxication is manifested by a distinct well-being and happy, cheerful humour. The cares and vexations of daily life recede into the background. A person becomes trifling, more accessible and amiable. Meanwhile irritability becomes manifest; he is provoked to abrupt changes of mood, is overbearing, gives himself up to outbreaks of temper and to fits of passion with grave excesses."

After the numbing effect of alcohol upon the sensorium has worn off, another affect makes itself felt. This consists in restlessness, the feeling that something is wrong, the sense of psychic

distress familiar to a large percentage of adults from their experiences next morning. It is difficult to find any one word to cover all the various manifestations of this feeling, but for want of a better term we may speak of it as apprehensiveness.

The primary and most fundamental symptoms of acute intoxication thus seem to be early psycho-sensory anæsthesia, and later apprehensiveness.

In the anomalous forms of intoxication we find usually a severe grade of disturbance of consciousness, usually accompanied with excitement, impulsiveness, and often criminal tendencies, but without serious paralytic effects. There may also be present distinct hallucinations and conditions of keen anxiety, which are otherwise foreign to intoxication. The two fundamental disturbances mentioned above explain for the most part the features of this group also, but in addition we have to reckon with a morbid predisposition which adds to the picture certain features of its own.

The restlessness, apprehensiveness, and irritability of the "sobering off" period are by no means the only manifestations of the painful affect. In certain cases we find, following severe or prolonged alcoholic excess, an outburst of terrifying hallucinations with corresponding delusions, the acute hallucinatory form of alcoholic psychosis. In these cases the orientation is usually retained, the morbid ideas and experiences are within the range of possibility, and do not come into any glaring contradiction with the actual situation. This requires but a brief mention, being a very pure form of psychosis, characterised by symptoms conditioned by the apprehensive state of the patient.

Chronic alcoholism is not regarded by the community at large as a psychosis, but the chronic alcoholic exhibits many psychopathic features. A mild psycho-sensory defect with euphoria determines the carelessness, improvidence, disregard of the requirements of morality and convention, the droll humour, over-valuation of self, and uncouth and overbearing manner. The apprehensive features commonly displayed are irritability, querulousness, jealousy, and suspicion. The alcohol, however, should not be blamed for all the manifestations of original depravity.

Of alcoholic stupor, Wernicke says: "The clouding of the sensorium in these cases, the somnolence, which is only paralleled

in cases of brain-tumour, may persist for weeks and even months, and makes the picture so similar to that of organic brain disease that for my part I have no doubt of the organic basis in hydrocephalus internus. Further, the disturbance of gait manifested by the patients as soon as they are out of bed resembles only that of senile hydrocephalus internus. The pulse too may be somewhat slowed, and general muscular rigidity with very distinct exaggeration of mechanical irritability is likewise common to these cases and to cases of senile hydrocephalus internus."

A few other forms of alcoholic psychosis require but a brief mention. Alcoholic paranoic conditions are gradually developed from long-continued intemperance, or may remain as the residual of otherwise recovered acute forms. The worst periodical drinkers show in many cases characteristic ear-marks of some constitutional tendency, either of the epileptic, hysterical, or manic-depressive type. Alcoholic pseudo-paralysis is characterised by deterioration, particularly in the field of memory and grasp, and by the prominence of physical signs. Alcoholic deteriorations will be discussed later.

In the polyneuritic psychosis (Korsakoff's psychosis) we find again, particularly at the beginning of the disorder, apprehensive symptoms in the way of restlessness, irritability, querulousness, sometimes a persecutory delusional trend and depressive hallucinations. But the feature which distinguishes this group is the defect in grasp of a special type, accompanied by euphoria. This defect is an impairment of retention, or rather of the ability to impress passing experiences upon the memory. Here again we find a familiar phenomenon. Though the psycho-sensory defect and euphoria are not identical with that of the first stage of intoxication, yet the analogy has led certain writers to suggest the inference that the defect in grasp is the direct result of the toxic action of the alcohol, while the apprehensive features are the result of poisons, elaborated in the course of disordered digestion and due to perverted metabolism. With the memory defect there exists in a certain percentage of cases a tendency to fabrication, analogous to that found in other similar conditions, for example, in senile psychoses. The loss of the connecting links with the past may render a correct time and place orientation impossible; but the patient grasps his immediate surroundings and reacts in a fairly normal manner thereto. In many

cases, however, the hold even on the immediate environment is a precarious one, and is entirely lost for short periods of time, during which the patient not only fills in the narrative of his immediate past with fabrications, but distorts his immediate impression, mistakes the place for some familiar locality, and, assisted by illusions and hallucinations, proceeds to engage in his accustomed business, or becomes the victim of imaginary fantastic experiences. In other words, we have temporary episodes of occupation-delirium.

The boundary line between these two conditions of polyneuritic psychosis and delirium is not a sharp one. For instance, one of our patients, though in general a fairly typical case of polyneuritic psychosis, yet persisted in believing that her child was somewhere about the place, saw it at times in the next bed, saw her mother among the patients in the yard, and had several episodes of a few seconds' duration, evidently provoked by neuritic pain and cardiac disturbance; during these she would manifest great anxiety, clasp her arms over her breast, believing herself to be holding her child, and seize the examiner's hand, calling him by the name of her husband. These states, which are really episodes of delirium tremens, are common enough in this psychosis, and show the close analogy of the two conditions.

In delirium tremens the defect in grasp is manifested by a failure of retention, the distortion of immediate impressions, and replacement of reality by fabrications, hallucinations, occupations, and imaginary fantastic experiences, due largely to the fact that the products of a lively imagination have come to have more reality to the patient than the testimony of the clouded sensorium. The apprehensive feature is seen in the colouring of most of the patient's experiences by anxiety and fear; for instance, the driver in his delirium makes desperate efforts to back his team out of all sorts of imaginary difficulties and dangers. Many of the hallucinations are also of a most terrifying content.

A characteristic feature is a certain grim humour, the Galgen-humor (gallows-humour) of the Germans. The patient, though perspiring with anxiety, views the situation in a facetious light, offers droll comments, and pictures in a joking way his hallucinations. Cheerfulness and deathly terror follow close upon each other. Bonhöffer regards the euphoria of the delirious patient as a sign of mental weakness. Kraepelin, however, takes another

view: "I have not been able to convince myself that it is confined to cases of psychic enfeeblement, and much prefer to ascribe it to the happy, care-free state which we observe in alcoholics generally, and in Korsakoff's psychosis especially, and which is also characteristic of simple intoxication."

It may be urged that delirium tremens is not really an alcoholic psychosis at all, but that the condition is provoked by some unknown factors, possibly disturbances of metabolism for which the alcohol is only a preparation. But that may be more or less true of the whole group. At any rate the symptomatic analogy between the different forms is strong evidence that the etiological factors are the same or very similar, whatever those factors may be.

To return again to the group as a whole, Kraepelin states the situation thus: "A glance over the forms of alcoholic psychosis just described seems to indicate the possibility of the development under the influence of that poison of two different groups of symptoms; on the one hand the delirious disturbances of retention and grasp, on the other hand the hallucinations and delusional formation. The first group of disturbances is present among the acute alcoholic manifestations and in simple chronic alcoholism; the latter group is not. This might suggest that in the former case we are dealing with the real and essential effect of the alcohol; in the latter case with the action of some intermediate factor." But it is difficult to see in this statement what provision has been made for the conditions of irritability, ill-humour, suspicion, etc., which are such prominent features throughout the entire group. Further, in our ultimate analysis of the cases, it is not so much the fact that the patient has delusions and hallucinations which is significant, as the fact that these latter have a definite character. In harmony with the patient's apprehensive state they might be depressing or terrifying; dependent upon the clouding of the sensorium, the hallucinations may be panoramic, or consist of familiar experiences; under certain conditions the ideas may be of an expansive character.

We find, then, in alcoholic psychosis two groups of symptoms, the first being conditioned by the apprehensive affect, and consisting of depression, restlessness, sleeplessness, irritability, anxiety, apprehension, suspicion, depressing or persecutory delu-

sions, depressing or terrifying hallucinations; the second being conditioned by psycho-sensory anæsthesia, and consisting of distortion of impressions, defective retention, euphoria, carelessness, fabrications, occupations, and fantastic hallucinatory experiences.

In regard to delusions and hallucinations of an expansive character, we might expect to find these dependent upon the euphoria of cases with memory defect. As a matter of fact they do occur: but the more pronounced instances of euphoria usually occur with rather grave loss of memory, and these patients are obviously not in a condition to develop and maintain a delusional system of any complexity. An expansive trend has been observed in a number of cases, associated with evidence of pronounced anatomical changes in the brain, such as unequal pupils and speech defect, but in which the examination of the spinal fluid has shown no lymphocytosis. In these cases the differential diagnosis from general paresis is a difficult problem. Another group presenting expansive ideas may be illustrated by the record of a case.

A. K., woman, aged 54, chronic valvular cardiac disease. Father was not entirely right, and paternal cousin was insane. Patient is very intemperate; she has had at least six previous admissions to this hospital, and has been in the workhouse a number of times. In her previous records we find statements like the following: Talks incoherently, despondent, noisy, abusive and assaulting—the doctors at B. Hospital have taken her heart out and mixed up her brain—tells a disconnected story to the effect that her husband gave her poison, tells of abuses at B., and how the ambulance tells a story about her. She has the physical symptoms of general paresis; she has delusions of great wealth and importance, thinks she owns a great deal of property, has millions of money in the bank, is one of the richest people in New York; she hears the voices of her dead relatives and sees them; says: "I have seen God in reality"; talked in a rambling strain, silly, confused, and excitable; noisy and excitable; says: "I have seen the Blessed Virgin and our Lord in the workhouse." At the workhouse she had delusions of persecution, of men entering her cell at night, fancied herself imposed upon by the other prisoners, whom she accused of conspiring against her. The above statements are selected from her various previous records. The delusions of persecution are strongly suggestive of

an alcoholic etiology, but the picture as a whole is not very typical of any psychosis. From each of the attacks she so completely recovered as to be discharged in her own custody. It must, however, be borne in mind that alcohol usually destroys the body before the mental symptoms have become so prominent as they are in this case. When a patient fifty-four years of age has had six previous attacks, the first being twenty-six years ago, has a bad cardiac lesion, and is yet alive to tell the tale, we must suspect a special vulnerability of that mind, a certain definite predisposition. What that predisposition is, is suggested by the course, which reminds us of manic-depressive insanity. From her record of her present stay in the institution we glean the following statements: Sings occasionally, seems alert, elaborates with all manner of details and rambles from topic to topic; her husband tried to kill her, she has about twelve million dollars left by her Uncle Sage, is one of Queen Victoria's daughters, "there she is behind you" (pointing to the nurse), is a relative of King Solomon, he was up at North Brother's Island. Her productions are well illustrated by the following: (Are you happy or sad?) "I feel happy, if it is a person that I like to see them near me, I never was happier than the four years I was a patient here. I could go to the laundry and go to bed, and if there was an entertainment I was invited, I never cared for entertainments, if I could have one of those little Bibles I could read, I didn't care about entertainments because I don't like them."

This production is significant. The alcoholic may be voluble on the subject of his delusions, but when a patient is voluble on all sorts of indifferent subjects, and pads out his narrative with all manner of irrelevant details, we usually find, as in this case, bad heredity, a history of previous attacks with complete recoveries, other manic traits, often delusions of grandeur; and the cases go on to recovery without residuals, as the case under consideration has done. A number of alcoholics with expansive ideas are of this type: the alcoholic manic will spin long yarns about his immense wealth the same as any other manic.

In differential diagnosis we must consider the fact that only a very small percentage of the population of our cities are total abstainers. In many families alcoholic beverages are upon the bill of fare more or less regularly. With the onset of a psy-

chosis, many formerly temperate persons begin to consume excessive amounts of alcohol, tea, coffee, or tobacco. With the development of a psychosis, many patients become also very intolerant of alcohol, and thus seem more intemperate than they really are. The relation of the patient's habits to the onset of the psychosis is important in determining to what extent intemperance is an etiological factor, and to what extent it is a symptom.

The differential diagnosis between certain forms of alcoholic psychosis and Dementia Præcox merits our special attention. If there is to be such a group as alcoholic insanity, we might hope to find something common to that group. These common features have been already pointed out. If we are to recognise any such group as Dementia Præcox, there should be also something common to that group. Kraepelin mentions as an important diagnostic point the loss of interest. In addition, there is a striking feature, which, if not common to the entire group, is at least sufficiently general to be of much assistance to us, viz. the absence of obvious cause and effect relations. Not but that cause and effect relations are operative, the same as everywhere else in creation, but they lie too deep for our present methods of psychologic analysis. For instance, the alcoholic is depressed for definite reasons; whether it is the persecutions of his neighbours, the infidelity of his wife, or the snakes under the bed, there are in any case definite and adequate reasons. The complaints of the precocious dement, on the other hand, are indefinite and incoherent, are a mere jumble of odd notions, cannot be written out in orderly sequence, are difficult of expression, unintelligible, often cannot be represented in thought by the normal individual at all, and are totally inadequate to explain the state of mind. In conduct the alcoholic may be resistive from suspicion, negativistic from sulkiness, assaultive upon provocation, real or supposed. The resistiveness and negativism of the precocious dement are in response to a blind and unreasoning impulse, for which the patient gives no adequate reasons. We must, however, differentiate obsessions, which are quite a different phenomenon, and distinguished by the fact that the patient has insight. This feature, the absence of adequate cause and effect relations, may be expressed as inconsistency or *incongruity*. If we then bear in mind as the common features

of alcoholic psychosis, apprehensiveness and disturbance of grasp, and the common features of Dementia Præcox as incongruity and loss of interest, we shall be in a position to size up our cases intelligently.

The record of a case is here presented by way of illustration.

B. M., aged 27, heredity negative. Had chorea as a child, later lived out; trifles disturbed her; at times would be so absorbed as not to listen to what was said to her; had few friends and no love affairs; was stated to be alcoholic. Was in this hospital six years ago and presented an atypical clinical picture: is said to have recovered completely. After that time she did not drink at all until lately. Six months ago patient gave up her place, went to church twice a day in order to save her soul. Two months ago was worried over her brother's drinking habits, and thought he was going to die. For the last month she has taken much alcohol. Informant sometimes gives her brandy to get her to sleep. Before admission she saw the Virgin; thought the devil was coming, and heard voices calling to her. In the hospital she said: "Oh, Jesus, Mary, and Joseph —Jesus, Mary, and Joseph—I don't want to be killed—I don't want to die—Jesus, Mary, and Joseph—Miss E., don't leave me." (Tell me what you are thinking about?) "I don't know how I feel—I feel pretty well—I am thinking of God and of Hell—I am thinking of—oh, I guess I will be all right." (What are your ideas?) "I am thinking I am going to die—I would like to make amends for my sins—I did not do very much sin—and I guess God will forgive me—I might have done a good deal. . . . I feel things, that is all—I feel things ahead of me —I cannot say what they are—I want to die and go to Heaven —if it is God's will I would like to live and be good—very good."

The following is an extract from the description of her conduct: It is almost impossible to get her to make an attempt to write. She gets up, looks anxiously about her, says: "Oh, Mary and Jesus," finally takes the pen in her hand and says: "Must I write—Yes! I will write—I can't write." She takes up the pen and lays it down two or three times, approaches the paper as if to write, then says: "I will not." After about ten minutes of parleying she writes her name. She is asked to write one of the test phrases, and the same performance is begun

again. Finally the attempt to get her to write is abandoned; she then becomes rather resistive, refuses to give up the pen, and says: "I will write." The paper is put away, and she reaches for it, puts it back in place, writes two words, misspelling them, then she begins to parley again. Similarly in the ward later, if asked to wait on table she offers to wash the dishes; if asked to make the beds she offers to polish the floor; ultimately does nothing. She has also committed assaults, for which no motive could ever be elicited. Her orientation is approximate as to many of the bare facts, but she shows her loss of the higher mental functions by her failure to grasp the significance of her situation.

Now it is impossible to analyse the wail of this patient and to state in definite terms what it is that is weighing on her mind. Her complaints are contradictory, vague, confused, and groundless. In her conduct she shows the conflict of ideas and impulses which has been much emphasised by the writers on Dementia Præcox. It is but a special manifestation of that incongruity discussed above. The patient is willing enough to co-operate, and yet for no obvious reason fails to do so. No explanation was given for her tendency to assault. Her inability to construct the situation seems to be dependent, not on defective grasp, but on failure of interest and attention. All this, with the fact that she seems to have been temperate until the beginning of the psychosis, and with several other suggestive features, establishes the diagnosis of Dementia Præcox. The alcoholic indulgence may have had something to do with the apprehensive state of the patient upon admission, although these states are common enough in Dementia Præcox. This view of the case is borne out by the subsequent course. Her apprehensiveness soon quietened down, but aside from that her mental state is the same as upon admission.

In sizing up these cases we have to make allowance for the extension of the process to contiguous fields. To take an example: as a result of the impairment of retention, there occurs a loss of memory, and then this amnesia may go back five or ten years before the beginning of the psychosis. Similarly a case of Dementia Præcox may show a primary loss of interest, and with that a profound loss of memory due to the failure to take notice. In many cases the reasons for the extension of the process are

much less obvious. Localised brain lesions are accompanied by psychologic phenomena impossible to account for. This emphasises the importance of studying the mechanism wherever that can be traced. In this connection special importance must be assigned to the initial symptoms of the disorder, and to the dominant symptoms at the time the patient is under observation.

Here the question might be suggested: Is not a case of Manic-Depressive Insanity or Dementia Præcox, provoked by alcohol, usually also coloured by alcoholic symptoms? That does not infrequently occur, though probably not as often as is usually supposed. The case usually shows a fairly clean cut picture of either one psychosis or the other. The explanation seems to be that if the mind has a predisposition towards some certain psychosis, it breaks down in that particular direction before the patient has consumed enough alcohol to give him very many of the characteristic alcoholic features. This reminds us of the observation that very few of the pathological changes due to alcohol are found post-mortem in hospitals for the insane as compared with the findings in general hospitals, due to the fact that these patients break down mentally before they have consumed enough alcohol to make its effects apparent in the parenchymatous organs.

The differential diagnosis of the Paranoic conditions requires but a brief mention. The term Paranoic condition is but a small part of a diagnosis, though it may be all that certain cases will justify. It simply states the fact that the patient has accumulated in some way a certain number of fairly coherent morbid ideas, but it is non-committal as to the nature of the process. Some Paranoic trends are developed in the course of chronic hypomanic states; sometimes we find that dream-like experiences have played an important part, and the process seems analogous to certain forms of Dementia Præcox. The differential diagnosis of the alcoholic Paranoic states, so far as it is possible, is to be made largely from the history of the development.

The mechanism of the development in a senile psychosis is practically the same as that of an alcoholic psychosis. The cases show the same defective grasp, the same affability, the same disorientation, the same fabrications, an analogous occupation delirium, and analogous persecutory ideas. The differential diagnosis of a doubtful case resolves itself into a rather unprofit-

able discussion of the question as to what extent intemperance has hastened the usual processes of decadence. In fact, alcoholic insanity seems to be a member of a larger group including in addition, senile psychoses, polyneuritic psychosis due to other causes, and certain traumatic cases. It is possible that these represent a special type of reaction, but more likely that the correlation between certain of the forms might be made upon anatomical grounds.

The relation of alcoholic insanity to Manic-Depressive Insanity and to Dementia Præcox is quite different. It may be that the symptomatic differentiation between certain cases of alcoholic insanity and certain cases of the Paranoid form of Dementia Præcox may be rather indefinite. Those cases offer a promising field for comparative study. But with that possible exception, it may be stated that the conditions are quite different. It is generally conceded that intemperance may provoke an attack of either Manic-Depressive Insanity or Dementia Præcox in individuals with a special predisposition, acting in the same way as any other debilitating factor. But the term alcoholic insanity is used to designate the manifestations of that poison in individuals of approximately normal mental make-up. The clinical picture is very definite, the condition is well entitled to recognition as a distinct disease-entity, the mechanism of the process is something fundamentally different from that of either Manic-Depressive Insanity or Dementia Præcox, and the conditions are not to be confused.

To recapitulate briefly: The symptoms of alcoholic insanity group themselves round two fundamental elements, apprehensiveness and defective grasp. In cases which do not yield to an analysis on this basis, some complicating factor must be sought. The symptomatology of the first stage of intoxication is very complex, but the primary disturbance seems to be defective grasp with consequent euphoria; the second stage is characterised by apprehensive symptoms. The anomalous forms of intoxication show the same features, and in addition the manifestations of a morbid predisposition. Acute hallucinatory insanity is a very pure form of psychosis dependent upon the apprehensive state of the patient. The polyneuritic form shows symptoms dependent on, or related to, both the fundamental symptoms referred to above, but is characterised as such by disturbance of grasp of a

special type. The chronic alcoholic shows symptoms of both series. The Paranoic states are gradually developed in chronic alcoholics, usually with the assistance of hallucinations, or are residuals of otherwise recovered acute forms. Alcoholic stupor is dependent probably upon anatomical changes in the brain. Periodic alcoholism is often the result of a constitutional disposition. Alcoholic deterioration is to be recognised by the character of its mechanism. Delirium tremens is closely allied to the polyneuritic psychosis, but represents a much more profound disturbance. An expansive trend may depend upon the euphoria associated with psycho-sensory anæsthesia; it occurs also in alcoholic pseudo-paralysis, and in alcoholic manics. Alcoholic insanity is analogous in its mechanism to senile psychoses, to the polyneuritic psychosis due to other causes, and to certain traumatic psychoses, but is fundamentally different from Manic-Depressive Insanity and Dementia Præcox.

DEMENTIA PRÆCOX.

By JOHN MACPHERSON, M.D.,
Commissioner in Lunacy for Scotland.

A GOOD deal of objection has been raised against the use of this term by Kraepelin, chiefly on the ground that Dementia has acquired a definite meaning, connoting, among other things, an incurable condition. It may be at once admitted that so far as concerns English terminology at any rate, the name is unfortunate, though by no means so confusing as is alleged. The use of the term in German gives rise to no embarrassment, for it does not conflict with the word "Blödsinn," the usual term for Dementia as we understand that word. It would perhaps be better for psychiatry in our own country if less attention were directed to the name, and a little more criticism devoted to the substance of the entity which Kraepelin has presented to us under the name of "Dementia Præcox." Is there such a clinical entity, and are its three sub-forms so intimately related as to justify their being classed together under that or any other name? These are, after all, the important questions to consider. If they can be answered in the affirmative, then we possess the most important advance in clinical psychiatry which has been

made in recent times. If they must ultimately be answered in the negative, the name is of small consequence, for it must pass away along with the erroneous idea which it denotes. Before passing from a consideration of the name, let me remark that it is not so wholly inapplicable as many persons would have us suppose. The condition as described by Kraepelin possesses, at any rate in its terminal stages, many points of resemblance to ordinary dementia; and 90 per cent. of the subjects remain permanently crippled in mind and go to form a very considerable proportion of the demented population of our asylums.

I have been frequently struck with the fact that among the many criticisms of Kraepelin's presentation there has been but a scanty attempt made to describe his main propositions in his own words. To do so at length would involve, practically, a translation of a long portion of his text-book, but I have resolved, in the interests of clearness and fairness, to present here an epitome of the basis upon which he founds his argument for the existence of the clinical entity which, whatever may ultimately be thought of it, is at present profoundly influencing current thought.

At the outset of his description, Kraepelin admits that the variety of clinical forms included under the generic term Dementia Præcox is very great, and that to a superficial observer the connection between them may not be readily perceptible. Yet there are certain basic symptoms underlying the more transitory appearances of the disease processes which all cases suffering from the affection possess in common.

(1) There is first the peculiar form of stupid listlessness from which one might conclude that the patients were oblivious to circumstances and events in their environment. On the contrary, however, the patients are able to appreciate and to, on occasion, describe everything that happens in their surroundings. They are able to tell where they are, to recognise individuals, and to count with accuracy. Only in profound stupor, and in the graver melancholic conditions, is orientation at all affected; yet in spite even of these serious mental disturbances the patients often remain clear in this respect. It is only when the thoughts of the patients are seriously affected by delusions that orientation and the perception of the surroundings are gravely interfered with.

(2) The sensory sphere is liable to grave implication, which often commences with dream perceptions. Hallucinations of the senses occasionally accompany the whole course of the disease; oftener they fade entirely, only to return with renewed force in the later stages of the disease. The hallucinations are at first of an unpleasant nature and very vivid. Later on they are regarded by the patients with more equanimity, and a few are even diverted by them.

(3) The consciousness of the patients, except in the excited and stuporose conditions, remains perfectly clear. More severe disturbances, however, not infrequently result in an affection of the faculty of attention, which is most apparent when an effort is made to sustain a conversation upon one topic for any time. On the other hand, it is not infrequent to see such patients observe stealthily what goes on around them, look curiously in at open doors, follow people at a distance, and manifest generally a sort of morbid curiosity in their surroundings.

(4) Memory is, proportionately, but little disturbed. The patients can, if they wish, give correct accounts of all past events in their experience, and even up to the time of the occurrence of the deepest dementia, retain all they have learned at school with astonishing tenacity. After severe stupor it generally happens that the memory is confused and weak, but that readily passes off in a few days or weeks.

(5) Sooner or later the thought processes of the patients always undergo deterioration. In milder cases this is shown merely in an increased abstraction of thought or in an unadjusted passing from one set of ideas to another. In more severe cases the affection manifests itself by incoherence of speech, with verbigeration or new word formation. But it has to be remembered that, in spite of these outward symptoms of thought disorder, the patients still preserve a fairly firm grasp of their surroundings, and that they can adjust themselves in a surprising manner to their environment. The presence of the peculiar phenomenon known as "Stereotypism" is almost always present as a symptom of the disordered thought processes. It results from the dominance of special ideas which for weeks and months at a time forcibly possess the mind, and frequently show themselves as repetition of senseless words, rhymes, or a strained play upon the superficial connection between words.

(6) The judgment of the patients is always without exception affected. They are incapable of acquiring new experiences, and so soon as they find themselves in novel and strange circumstances they become helpless. They are unable fully to comprehend that the change is in themselves and not in their surroundings, consequently they form erroneous and false conceptions of their position.

(7) Upon this basis, delusions of a fleeting or lasting nature are apt to arise. In the early stages of the disease the contents of the delusions are sad or hypochondriacal. Later on, grandiose ideas begin to appear, which may gradually become dominant. As a general rule, and owing to the rapidly developing mental weakness, all forms of delusion exhibit a nonsensical, quixotic stamp. They are, moreover, not constant, but change their character from time to time in accordance with the mental condition of the subject. In the Paranoid form of the disease the delusions present a more lasting character, but even there they ultimately become incoherent and dissociated. The tendency for all these delusions is ultimately to disappear in forgetfulness.

(8) The mental disposition undergoes marked change. At first the patients are depressed, which condition is varied by outbursts of excitement or of unrestrained joviality. Next follows a state of good-natured dementia, which is accompanied by a peculiar listlessness, a loss of affection for relatives and friends, and a disinclination for employment or recreation. They seem to experience neither pleasure nor pain, to exhibit neither desire nor apprehension, but live for the day only in a dull and stupid manner. They also manifest indifference towards bodily discomfort and slight pain; the only interest or pleasure that remains is that for food. Towards the later stages of the disease a condition of complete listlessness supervenes, varied occasionally by outbursts of irritability.

(9) The behaviour of the patients adds importantly to the clinical picture. They lose all power of initiative and neglect their work and their obligations. Along with this loss of initiative, there is displayed passing or lasting impulses towards suicide. The impulse to self-destruction does not imply any increased initiative on the part of the patient, but is only a result of a motor excitement which impels to destroy. These impulses are aimless, and are due to the sudden relaxation of the previous

inhibitory tension under the influence of excitement. All the impulses are equally aimless, such as the breaking of windows and dishes, as well as attempts at self-mutilation. All the common actions of the subjects, such as walking, writing, eating, hand-shaking, are apt to be traversed and interrupted by impulses. In this way are exhibited those mannerisms and peculiarities which are so characteristic of the patients.

(10) To this affection of the will power, coupled with the prevalence of automatic impulses, is attributable that peculiar negativity which manifests itself as an obstinate opposition to every change of position, to taking food, to putting on or off clothing, etc.; to the same cause is due the retention of urine, fæces, and saliva, the creeping under beds, the refusal to answer questions, the "touchings," and the general inaccessibility to all attempts at approach on the part of others. This negativity which can seldom be influenced from without, may yet through some internal impulse be suddenly counteracted and the patient may all at once engage in some useful or senseless piece of work, only, however, to fall back again into his former immobility.

(11) The working capacity of the patients is without exception lowered and may be seriously affected. Their work has to be carefully supervised, for they stop at the slightest difficulty, and should they meet with anything novel or unusual, they are incapable of adapting themselves to the altered condition. In the performance of artistic work, to which they may have been previously trained, they manifest along with, it may be, great technical ability, a failure in the sense of beauty and proportion, and a decided inclination towards peculiarities. In musical patients the decay of the finer artistic feelings is early manifested, and exhibitions of a distorted and capricious nature are the general rule.

In addition to the above list of mental symptoms, which are common to all forms of Dementia Præcox, Kraepelin mentions a series of bodily symptoms which he admits have not been established and correlated, but which are sufficiently important to be briefly detailed.

(1) In about 18 per cent. of all cases he has observed the occurrence of epileptiform or tetanic spasms, or even of apoplectiform attacks followed by more or less transient paralysis. All

such attacks are twice as numerous in the female as in the male sex.

(2) Choreiform movements of a special and lasting character have been repeatedly observed.

(3) The tendon reflexes are regularly exaggerated, often markedly so; there is also often an increase in the mechanical excitability of the muscles and nerves.

(4) The pupils are often widely dilated, especially in the excited stages of the disease; here and there one observes a difference in the relative sizes of the pupils.

(5) The heart's action is subject to great irregularity, sometimes slow, often accelerated, at other times weak and irregular. Vasomotor changes of all kinds are common.

(6) The sweat and salivary secretions are liable to be greatly increased in certain patients and at certain stages of the disease.

(7) Diffuse enlargement of the thyroid gland has frequently been noted prior to the onset of the mental symptoms, and it has been noted to return quickly at the circumference of the gland during the development of the malady. In a few cases distinct exophthalmos was present. Finally, among the histories of the relatives of the patients it is not uncommon to find myxœdematous symptoms.

(8) The sleep of the patients is invariably disturbed.

(9) The body weight, irrespective of the quantity or quality of the food taken, is liable to great variation, depending upon the recurrence and subsidence of the acute attacks. When permanent dementia ensues and the acute attacks cease, it is usual to find the patients take on flesh and present a well-nourished appearance.

The three divisions of Dementia Præcox, according to Kraepelin, are: Hebephrenia, Katatonia, and Dementia Paranoides. He defines each shortly as follows: "By Hebephrenia is meant all those forms of Dementia Præcox in which a uniform, more or less profound condition of mental weakness is developed under the accompanying influence of subacute—more seldom of acute —mental disturbances."

"Katatonia is essentially a peculiar disease process progressing through stupor or excitement to a condition of dementia, with intercurrent symptoms of Negativism, Stereotypism, and Auto-Suggestibility in the spheres of action and work."

"Dementia Paranoides includes a group of cases in which delusions and hallucinations are produced in an otherwise clear mind throughout many years of a steadily advancing mental weakness."

That these divisions are largely artificial is candidly admitted by the author (*vide* "Lehrbuch," pt. ii. p. 148, 6th ed.). "There can," he says, "be no rigid line drawn between the divisions, because the cases run into each other and overlap continually. There are a whole series of recurring forms, but between them lie such a variety of transition cases that, in spite of all effort, it is impossible to classify each case, without exception, in a given category."

In forming an estimate of Kraepelin's conception of Dementia Præcox, as I have attempted to present it above, largely in his own words, it is necessary to approach the subject from the point of view of the author and of those who agree with him, Up to within the last two decades of the nineteenth century the progress of psychiatry was somewhat slow and shadowy, especially in the field of pathological research. The great pathological advances of the last twenty-five years have not as yet very markedly affected the clinical side of the study of mental diseases. Consequently classification is chaotic. There may not be much harm in that so far as individual physicians are concerned, for long practice endows the physician with an empirical knowledge, which, however, perishes with himself. The great want has been a nomenclature or language by means of which each earnest, educated physician can communicate his empirical impressions to others. This Kraepelin has done effectively in this instance. It may be that the language, or perhaps I ought to say the words, are uncouth, but the idea underlying them is a living reality. Out of the unutterable confusion of psychiatric nomenclature Kraepelin selects a group of cases—not a large group, only about 15 per cent. of the total annual admissions to his Clinique—he predicates certain things regarding their symptoms, diagnosis, and prognosis, and asks his brethren throughout the world to criticise his position. These patients were previously unattached, so to speak; each man classified them variously according to his own peculiar views, with the result that our knowledge was becoming hazy in respect to the prognosis of various types of acute insanity.

The great importance, then, of Kraepelin's presentation of

Dementia Præcox lies in the essential advance he has attained in the prognosis of a hitherto unclassified group of cases. In this group a progressive mental weakness is the constant and leading feature. But progressive mental weakness alone does not constitute Dementia Præcox. There are other symptoms which enable the physician from an early stage of the disease to form an unfavourable opinion as to its course. These symptoms, which have already been detailed, are Negativism, Peculiar Mannerisms, Stereotypism, Impulses (Willensstörung), and especially Disorders of Judgment (Urtheilsfähigkeit). Strongly confirmatory of Kraepelin's position is the fact that these symptoms are common to a large number of so-called terminal dements.

It is an objection of considerable force that a certain proportion of the group make satisfactory recoveries—8 per cent. according to Kraepelin himself. The only possible answer is that our knowledge is not sufficiently advanced to enable us to distinguish allied types which are not really cases of progressive dementia from those which are.

Some objection has been raised on the ground that the conception is neither pathological nor etiological. This is a futile objection when our knowledge allows us to supply neither the one nor the other. The classification is at any rate thoroughly clinical, and its merit depends upon the valuable aid it affords in prognosis, which is, after all, the most important detail in psychiatric medicine at the present day. Should it be established that these affections, or some of them, depend upon a toxic basis, as Dr Lewis Bruce hopes to be able to substantiate, we shall not even then have fully solved the mystery. It must be remembered that in many instances Dementia Præcox is a slowly progressive disease, extending over many years; that it often occurs in individuals who have for some reason failed to develop properly either mentally or physically; and that its commencement may be insidious and unobserved. We must look, therefore, for some other reason than a toxic causation alone. Keeping in mind Kraepelin's observations upon the changes in the thyroid gland in some of his patients, as well as other facts in development, it is not unlikely that physiology has yet much to teach us regarding the influence of various bodily glands upon nervous mechanism.

In concluding these remarks, I may perhaps be pardoned for

pointing out that the opponents of Kraepelin's conception of Dementia Præcox write and speak as if it were put forward as an ultimate solution of the problem of psychiatric classification which threatens to darken counsel for all time coming. This illusion of finality may have its uses in many departments of life, and even in some of the more exact sciences; but it is surely misplaced when applied to psychiatry, which is now in a state of flux and change in all its branches. The truth is that Kraepelin himself would be the last man to assert that his conception was more than tentative—except, of course, as regards diagnosis and prognosis. He has handed on the torch and even trimmed it. His very errors will form a necessary step in the progress of our knowledge. In the meantime many of the leading alienists in both continents have accepted his presentation as a simple, workable advance, the outcome of genius and of incalculable patient observation.

NOTE.—The Editors have requested that I should, for the benefit of the non-psychiatric reader, explain some of the unusual technical terms which occur in this paper. I gladly do so with apologies for their use.

Hebephrenia (ἥβη, puberty; φρήν, mind), lit., the mind of youth.

Katatonia (κατατείνω, I stretch tightly) is applied to this disease because of the cataleptic rigidity which frequently accompanies its stuporose phases.

Dementia paranoides. Paranoia (παρα, beyond; νοέω, I know) is a term now usually applied to delusional insanities.

Stereotypism is a term applied to designate the expression of recurrent phrases which are the outward signs of certain fixed ideas which dominate, for longer or shorter periods, the minds of the patients.

Verbigeration means the repetition of sounds or words, often meaningless words, invented by the patient himself.

Negativism implies a state of chronic resistance on the part of the patient to conform to the necessary conventions of life.

Auto-suggestion is used to describe the presence in the mind of obsessions or independently arising suggestions which precede and lead up to the innumerable impulses in action which are a leading feature in the symptomatology of the disease.

Abstracts

ANATOMY.

ZUR ANATOMIE DER FISSURA CALCARINA. E. ZUCKERKANDL,
(85) *Arb. a. d. neurolog. Institut a. d. Wien. Universität*, Bd. 13, S. 25.

ZUR ANATOMIE DER UEBERGANGSWINDUNGEN. E. ZUCKER-
(86) KANDL, *Ibid.*, S. 141.

BOTH these papers are devoted to description of the minute morphology of these cerebral fissures and gyri in the lower and higher apes and in man. As such, suitable abstracts cannot be easily made of their contents, and those interested in the subjects must be referred to the original articles.

It may be noted, however, that in the first paper annectant gyri (G. cuneo-linguales) are described in the calcarine fissures of some of the higher apes, though Cunningham has regarded them as peculiar to the human brain.

In the same paper the question is discussed in how far the F. calcarinæ of the simian and human brains are homologues. According to Cunningham the F. calcarina of the monkey corresponds to the anterior limb of the calcarine fissure of man, while Elliot Smith believes it is represented only in the posterior limb. Zuckerkandl rejects both these views and regards the human and simian calcarine fissures, each as a whole, as true homologues.

GORDON HOLMES.

THE COMPARATIVE ANATOMY OF THE CENTRAL CANAL.
(87) **(Vergleichend-anatomische Untersuchungen über den Bau des Zentralkanales bei den Säugetieren.)** P. BIACH, *Arb. a. d. neurolog. Institut a. d. Wien. Universität*, Bd. 13, S. 399.

THE central canal of the spinal cord has been investigated by the author in representatives of almost all the mammalian classes. He finds that in the lower mammals, in contrast to man, it is almost always elliptical in cross-section, its greatest length lying in the dorso-ventral axis of the cord. It is always exactly in the centre of the lateral diameter of the cord; in the cervical segments it generally lies in the centre of the commissure, but in the dorsal and sacral segments in the ventral half of the commissure, owing

to the increased breadth of the dorsal half of the latter. In almost all classes (the natantia are an exception) it has a distinct lumen which contains a granular, felt-like, or homogeneous material, in which cells of glial or ependymal origin or even leucocytes are often embedded. In many specimens a homogeneous structure, round on cross-section, was found within the canal. This is the structure which has been described as Reissner's fibre, but Biach agrees with Studnicka that it is not a nervous structure, but probably only a coagulated deposit within the canal. The ependyma that lines the canal generally consists of a single layer of cubical cells, from the dorsal and ventral surfaces of which bundles of glial fibrils radiate into the substance of the cord.

The substantia gelatinosa centralis has no definite boundaries. It consists of a dense network of neuroglial fibrils, and nerve cells and fibres are frequently found within it. Its size is very variable in the different orders. In some of the lower mammals it extends along the median border of the dorsal horns to become continuous with the substantia gelatinosa Rolandi; in other orders in which there is no continuity between them their identity is indicated by the presence of similar cells in each. Besides the few myelinated fibres which extend into it from all sides, the substantia gelatinosa centralis almost invariably contains in the lumbo-sacral segments small bundles of longitudinally running fibres which can be followed into the tip of the conus. GORDON HOLMES.

ON THE INTRABULBAR CONNECTION OF THE TRIGEMINUS WITH THE VAGUS. (88) **(Ueber die intrabulbaren Verbindungen des Trigeminus zum Vagus.)** M. GROSSMANN, *Arb. a. d. neurolog. Institut an Wien*, Bd. 13, 1906, S. 194.

FROM his experiments on the effect produced on the heart by stimulation of the central end of the V., and on the effect of the nasal reflex on the circulation and respiration, the author came to the conclusion that there is a direct functional connection between the V. nerve on the one hand, and the vagus and phrenic nerves on the other. Stimulation of the central end of the V. raises the blood pressure and influences the rhythm of the heart and the movements of the diaphragm.

A direct anatomical connection between the trigeminus and the vago-glosso-pharyngeal nuclei has been already described. Wallenberg found, in a case in which the sensory root of the fifth was degenerated, a special bundle lying in the dorso-medial angle of its spinal root which could be followed into the tractus solitarius; and Bruce and Hagelstamm found changes in the latter after

degeneration of the sensory root of the trigeminus. Cassierer and Schiff found this bundle intact despite destruction of the IX. and X. nuclei, and were able to trace it upwards to the sensory nucleus of the V. Lewandowsky has denied the existence of any such connection between these two nuclei, as he was unable to discover it in his experiments on monkeys.

The author's experiment consisted in the removal of the Gasserian ganglion in a monkey and the subsequent examination of the brain-stem by Marchi's method. He obtained complete degeneration of the sensory V. root; some of its fibres entered the cerebral root, others terminated in the motor trigeminus nucleus, but the majority ended in the sensory nucleus and in the spinal root. No direct root fibres passed to the cerebellum.

At the caudal end of the sensory nucleus a small collection of cells, to which the name nucleus ovalis is given, was found lying at the dorso-medial angle of the spinal root and its substantia gelatinosa Rolandi. It seems to be a continuation of the sensory nucleus, and its cells resemble those of it rather than those of the substantia Rolandi. It is surrounded by degenerated fibres, some of which end in it and others pass to it from the spinal root. Further caudalwards it moves dorsalwards and lies embedded in the ventro-medial angle of the spinal root of the vestibular nerve, and finally it joins the substantia gelatinosa of the fasciculus solitarius. It is accompanied for its whole length by degenerated fibres of the trigeminus.

These fibres and the nucleus ovalis then represent a direct anatomical connection between the trigeminus and the vago-glosso-pharyngeal nerves. It has been assumed (Roller, Wallenberg, Edinger) that the fasciculus solitarius and its cerebralward projection belong to the function of taste, but the investigation of Cassierer and Schiff's case disproves this view, and Grossmann adds that as fibres from all three divisions of the trigeminus enter it the nucleus ovalis cannot be only a taste centre.

GORDON HOLMES.

ON THE SENSORY ROOTS OF THE MEDULLA OBLONGATA IN MAN.

(89) **(Beiträge zur Kenntnis der sensiblen Wurzeln der Medulla oblongata beim Menschen.)** E. HULLES, *Arb. a. d. neurolog. Institut a. d. Wien. Universität*, Bd. 13, S. 392.

THE author has attempted to determine how far neuroglia extends into the roots of the sensory cranial nerves. When the vagus root is cut longitudinally as it enters the medulla it is seen that there is an interruption of its myeline sheaths a short distance before its entry; over a zone about 1 mm. broad its fibres are not stained by

Weigert's method. This pale band is convex peripheralwards, and by suitable staining it is seen that it is the region where the root fibres lose their sheaths of Schwann, and the peripheral limit of the extension of the neuroglia of the bulb along the root. In some of the nerves (V. and X.), however, irregular projections of neuroglia extend further along the nerve. The dense band of glia which occupies the area that stains palely by Weigert's method is continuous with the marginal zone of neuroglia of the central nervous system which extends over the most proximal portion of the root as a sheath. The pia mater surrounds the whole and joins the connective tissue sheath of the nerve.

This condition is compared with what Levi has found in the posterior spinal roots. The chief difference is that in the cranial nerves the band of neuroglia lies further peripheral and is broader than in the spinal roots, and that the latter are not surrounded by a sheath of pia mater.

Hulles also examined the intramedullary portion of the sensory roots by Bielschowsky's method in the medulla of newborn babes. He failed to find bifurcation of any of the fibres of the vagus, but saw a few fibres of the trigeminus branch after they had entered the pons. GORDON HOLMES.

THE MODE OF CONNECTION OF THE MEDULLATED NERVE FIBRE WITH ITS CELL BODY. (90) OLIVER S. STRONG, *Journ. of Comp. Neur. and Psych.*, Vol. xvi., No. 6, Nov. 1906, p. 397.

THIS paper gives some demonstration of the subject title as studied in the lumbar cord of an infant, by various modifications of the Weigert-Pal method.

The "axone cone" emerging from the cell body may either taper regularly, or first gradually and then more abruptly, to the narrow neck of the axis-cylinder process. This attenuation of the axone at a point near the cell body has long been known, and may be partly an artefact, but is probably not entirely so, as it has been demonstrated by so many methods. Beyond it the axone expands and at the same place becomes medullated; the expansion and myelination seem to be correlated as regards whether they occur abruptly or gradually.

Only one collateral branch was observed from the naked part of the axone, but, as the author remarks, this cannot be regarded as negative evidence of much weight, for the methods employed were incapable of demonstrating very fine processes. For this reason the axones of the smaller cells were not available for study.

J. H. HARVEY PIRIE.

THE DEVELOPMENT OF THE VERTEBRATE NERVE-CELL: A (91) CYTOLOGICAL STUDY OF THE NEUROBLAST-NUCLEUS.
JOHN CAMERON, *Brain*, 1906, p. 332.

THE main aim of this paper is to emphasise the importance of recognising that the nuclei of embryonic nerve-cells (neuroblasts) are centres of great metabolic activity. One of the earliest indications of the latter is shown in lower vertebrates (*e.g.* fishes and amphibians) by an ingestion of yolk-particles at one definite pole of the nuclear wall. For this region the title of *assimilative pole* has been adopted. The ingested yolk is elaborated within the nucleus, and stored up as chromatic material, which first shows near this pole.

Still more striking evidence of the existence of this nuclear metabolic activity is shown in these lower vertebrates by the fact that part, at least, of the earliest *rudiment* of the axis-cylinder process arises in the form of successive extrusions of nuclear achromatic substance from the assimilative pole of the neuroblast-nucleus. This pole thus becomes the *axon-pole*. The assimilative or axon-pole of the neuroblast-nucleus has always a definite direction. Thus it looks towards the ventricular cavity in the cerebral hemispheres, and forwards as well as outwards in the anterior cornua of the spinal cord. It is found, further, that not only the axon-rudiment, but also a considerable proportion of the cytoplasm of the future nerve-cell is produced from the neuroblast-nucleus, owing to the continued exercise of this *achromatin function*.

The material which is extruded is achromatic only in the *nascent* condition. It very soon, however, undergoes a characteristic process of *rechromatisation*, which affects the extruded perinuclear substance in a definite manner, and gives rise to the neuro-fibrillary network, and probably also to the Nissl bodies.

It is a significant fact that the neuroblast-nuclei exhibit structural changes as evidence of their metabolic activity. The most striking is a disappearance of the accumulated chromatic material from the axon-pole, which occurs during the period that the axon-rudiment is being given off. The chromatic element is thus retransformed into nascent achromatic element, in which condition it is discharged from the nucleus.

The results of this research go to show that three types of neuroblasts may be recognised. For these the names of α, β, and γ neuroblasts have been adopted.

1. The α neuroblasts retain their embryonic character more or less. They probably form a reserve of young nerve-cells.
2. The β neuroblasts become invested by a very scanty cytoplastic envelope.
3. The γ neuroblasts become transformed into actively functioning nerve-cells.

It is important to note that nerve-cells begin their life history as the *a* type of neuroblast, and pass successively through the stages represented by the β and γ types.

Several observers, such as Held, Rohde, Holmgren, Scott, etc., have shown that the cytoplasm of nerve-cells is rich in nuclein compounds. This seems to suggest that a part of it at least is derived from the nucleus, as shown in this research. Fragnito and Capobianco go so far as to declare that the major portion of the adult nerve-cell cytoplasm is derived from the disintegration and fusion of the nuclei of neuroblasts which they style *secondary neuroblasts*, while the nuclei of other neuroblasts (called by them *primary neuroblasts*) persist as the adult cell-nuclei.

The nucleus not only gives part origin to the cytoplasm of the nerve-cell, but also furnishes a supply of fresh material to replenish the latter, when occasion demands, throughout the life history of the individual. This function has been clearly demonstrated by a study of the effects of fatigue upon the nerve-cells, as investigated by, among others, Hodge, Lambert, Mann, and Vas. The nucleus is thus to be regarded as the *nutritive centre* for the nerve-cell. As a result of this, it is obvious that a modification in our present acceptation of the Wallerian law of nerve degeneration will be necessary. The latter would thus come to read somewhat as follows: An axon, when cut off from its nutritive centre, *i.e.* the *nucleus* of a nerve-cell, soon exhibits degenerative changes.

AUTHOR'S ABSTRACT.

ON NUCLEAR DIVISION IN THE ANTERIOR HORN CELLS IN MAN. (Über Kernteilungen in den Vorderhornzellen des Menschen.) ORZECHOWSKI, *Arb. a. d. neurolog. Instit. a. d. Wien. Universität*, Bd. 13, 1906, S. 324.

(92)

THE author describes and figures certain appearances observed in the cells of the anterior horn of the spinal cord after amputation of the forearm. The patient (aged 51) whose cord was examined died fifteen days after the operation, which was performed on account of the presence of tubercle. Before death there was slight elevation of temperature. The cord was fixed in formalin. The lower cervical and upper two dorsal segments were cut in serial section, and the sections stained with thionin. With the exception of the changes in the nerve-cells, little pathological alteration had taken place in the spinal cord. On the side of the degeneration, all the groups of nerve-cells, except the ventro-median, exhibited signs of degeneration. A number of nuclei of anterior horn-cells were observed to be in process of division. In such dividing nuclei, degenerative changes could frequently be

seen. Many nerve-cells, including some in which nuclear segmentation could be observed, and others with unchanged and undegenerated nuclei, presented an appearance of complete chromatolysis. The author describes the cell changes as post-resective appearances complicated by an inflammatory process spreading from the seat of amputation along the nerve-sheaths.

The spinal cord was somewhat flattened in the ventro-dorsal diameter, and the majority of the flattened motor cells lay with their long axes frontally situated. The nuclear figures, of which about forty were observed, are contained almost always in such spindle-shaped cells with short ventro-dorsal and long frontal diameters. Within these cells the nuclear figures lie with their long axes approximately ventro-dorsally placed, forming an acute angle with the sagittal line. The appearance presented in most cases is that of an elongated, deeply-staining figure, consisting of two daughter nuclei united by an intervening bridge of less deeply-staining nuclear substance. Around the figure lies a clear area which is considered to arise from the fluid part of the nuclear substance after solution of the nuclear membrane. In some cases the daughter nuclei are well marked off and connected to each other merely by threads of nuclear substance, and the process has occasionally advanced to almost complete separation of the nuclei. A few of the figures appear to indicate division into three or four daughter structures. The nuclei frequently contain small granules united by fine threads. The presence of degenerative changes in the cells raises the question whether the figures evidence actual division, or are results of degeneration which simulate division. The author considers that degeneration has set in in the course of a progressive regenerative process, but that the figures do not owe their origin to degeneration. It is possible that the structures which are undergoing division are not nuclei but nucleoli. Many of the appearances have a superficial resemblance to karyokinesis, but the division which is seen in the majority is neither of the direct nor of the indirect type. The explanation of this divergence from the recognised forms is to be sought in the morphology and chemical constitution of the nuclei of the large nerve-cells.

The author discusses the bearing of his results upon the general question of the possibility of proliferation of nerve-cells. He has been unable to demonstrate instances of complete separation of daughter nuclei, but cannot conclude from this that, had the spinal cord been examined at a later period, such a condition would not have been observed. W. A. JOLLY.

PSYCHOLOGY.

BRAIN AND THOUGHT. (**Cerveau et pensée.**) ALFRED BINET, (93) *Archiv. de Psychol.*, Aug. 1906, p. 1.

I. THE connection of the brain with thought is the tangled skein of every philosophical system. Parallelism is the working hypothesis which dispenses most with the Chinese puzzles of metaphysics. Its dominant contention is that between the neurological phenomena and the mental there exists only a rigorous simultaneity in time. But parallelism keeps dangerously near the notion of causality, and finally loses itself in materialism. Nor are its premises unimpeachable. Berkeley and succeeding idealists took the other way of escape, and postulated the purely verbal solution of the identity of brain with the objects of perception.

II. The cardinal difficulty of the discussion consists essentially in the irreconcilability between the current produced in the cells and fibres of the nervous system and the perceptions belonging to psychical life. When an external object excites our senses, the hang of the mechanism is much the same as the transmission of the full blast of an orchestra across a telephone wire with a metal diaphragm of a few millimetres in diameter. It is not the sound which is propagated along the wire, but its transformation into electricity. The size of an object (which is a source of comic embarrassment for realists, who want to know how a star and its properties can be brought to heel by a telescope and lodged in the brain) is a negligible difficulty when size is taken to mean the duration of the excitation, or its intensity, or the number of fibres it intersects, or all these qualities at once. Objects are pure phantoms created by our imaginations to explain the linkage or the genesis of our sensations. Refusal to admit that the little brain may contain the great universe is due to making a fetish of mechanism. Mechanical continuity is only a symbol, helpful but unessential.

III. The results of the author's observations on the relations between consciousness and the nervous system are embodied in various propositions.

1. Considered as a physical object, the nervous system is a closed and self-sufficing system.

The brain takes in raw materials and turns out finished goods, just like a machine in a factory. Our most poignant joys and sorrows are a super-added encumbrance, as useless to the brain as to the liver and kidney.

2. If consciousness, abstracted from what constitutes its object, is not a material object, a material phenomenon, a material force, nor any constituent whatever of the material world, it is useless to

seek how contact is made between consciousness and that fragment of matter which constitutes the brain.

Consciousness may be compared to a light bathing the interior of the nervous substance, or to an atmosphere in the midst of which nervous movements are produced.

3. The objects we perceive being, as we have seen, of material nature (for every sensation is mixed, at once material and mental), the relation of these objects with our brain, which is equally of material nature, ought to be describable and even observed.

I see an object: is it this image which is going to act on my muscles, or is it the motor current? If it is the image, the motor current is useless. If it is the motor current, where does the image come in?

4. Although the question of the relations between brain and thought is the authentic contemporary presentation of an older question, that of the connection of the soul and the body, it seems evident that this problem is clearly distinct from the problem of the correlation of the mental and the physical.

The problems are akin, for the brain is a material body, and thought includes what is knowable of the soul. But suppress the second problem, you do not get rid of the first. Suppose brain and thought to be similar in make-up, both physical or both mental, you will have just as much trouble in establishing their interaction. Let the pyramidal tract be physical or mental, it is equally difficult to explain the action of the will on it.

5. The whole group of psychical phenomena (sensations, perceptions, ratiocinations, etc.) is localised outside of the nervous system.

These emotions and these efforts are the perception of certain muscular or visceral states outside the brain. For the man ignorant of anatomy the brain has no existence, and the facts of conscious life fill the whole cycle of events from excitation to response. For all of us at some period of our life the picture of the universe is made up of objects perceived plus a series of special phenomena like the emotions and the will. Put the nervous system into its proper place and its intercalation makes no difference in the interpretation. Even the products of the mind communing with itself are essentially excitants of the nervous system. Take a recollection. It represents a previous perception; it is its footprint; everything runs on as if the excitant were still active. The excitation is renewed twice, that is all. It is none the less external to the brain.

6. The brain conditions and standardises our perceptions.

Perceived objects which excite a nervous system have need of that nervous system, not for their existence, but merely for their perceptibility. Just as light does not create the objects it

illuminates, so consciousness creates neither the brain nor the external world. The external world is the finger which is ready to press on the button of the electric light apparatus; the button and the whole electric installation is the brain; the light shoots out and illumines objects which existed before it, and could continue to exist after its extinction. The brain is the standard, and our perceptions for consciousness only seizes, amongst all the knowable properties of an object, those which have traversed the nerve. The existence of hallucinations where a special state of the nervous system engenders perceptions in the absence of a corresponding external object shows that consciousness is shut up in the nervous system.

7. As the intermediate link in the relations between consciousness and the external object, the brain belongs to the group of unknown quantities and not to the group of sensations.

Appearance must be distinguished from reality. Physiologists have shown that the quality of each sensation depends on the nature of the nerve excited and not of the stimulant which excites it; the optic nerve makes the light response always, whether tested by a luminous ray, an electric current, or a blow. There is no need to push the induction that light may not exist outside our brain; from the moment that the existence is granted of an intermediate link in the chain of our perceptions, no limit can be placed to its possible influence. Appearance is sensation, reality is an unknown quantity. Every object can be thought of in two ways: as a sensation, or the appearance it assumes in our perceptions; or as X the unknown cause of our sensations. These are, as it were, two planes, in which the same object can be localised. But it is illegitimate to place ourselves in both planes at once. This is what we do when we say the brain is the producer of thought. The true cause of conscious life is the X brain, unknown and inaccessible, whose relations with our thought we cannot seize, since we do not perceive it, and know nothing of its structure.

J. Roy Tannahill.

PATHOLOGY.

DEGENERATIVE NEURITIS AND ATROPHY OF THE AUDITORY

(94) **NERVE. (Weitere Beiträge zur Kenntnis der degenerativen Neuritis und Atrophie des Hörnerven.)** Wittmaack, *Ztschr. f. Ohrenheilk.*, Bd. 53, H. 1, S 1.

This paper is a sequel to one formerly published by the same author (Bd. 51, S. 161) on experimental degenerative neuritis of the auditory nerve, and contains an account of histopathological

examinations of the organ of hearing in two cases of concomitant neuritis due to scarlatina, one of auditory atrophy after typhus, and one case of degenerative neuritis in each of the following conditions: senile decay (in a dog), diabetes mellitus, and senile cachexia (tuberculosis). Short clinical notes as well as the results of the functional examination are given in several of the cases.

The following conclusions may be drawn from these investigations: Atrophy of Corti's organ is secondary to neuritis which involves the cochlear nerve, ganglion cells, and nerve filaments; pathological changes begin in the periphery of the nerve and reach their highest degree in the modiolus cochleæ; in severe cases there is great increase in the interstitial tissue, but in almost all cases there is reduction of nerve bundles and also of cells in the spiral ganglion: the latter show crenation of the cell contour, vacuolation of protoplasm, disappearance of Nissl's spindles, and shrinkage of the nucleus; the nerve filaments show ampullation of the medullary tubes, and, in the later stages, the medullary sheaths can no longer be recognised.

1. In the mildest forms of neuritis Corti's organ shows shedding and degeneration of isolated hair cells, often combined with adhesion of the membrana tectoria to the papilla basilaris; the supporting apparatus is well preserved. This condition is seen in slow progressive or mild forms of degeneration, and as a rule only produces slight degrees of deafness.

2. The second degree is characterised by the shedding of a greater number of hair cells, with slight flattening of the supporting apparatus. This change is found in moderately severe acute lesions of the nerve.

3. The most severe degree of involution of Corti's organ consists in the metamorphosis of the papilla basilaris into an almost flat epithelial layer, in which neither the hair cells nor the characteristic structure of the supporting apparatus are recognisable. This condition has only been found by Wittmaack in the most severe and quickly progressing forms of degenerative neuritis. In both 2 and 3 the membrana tectoria is atrophic and adherent to the papilla basilaris.

The great difference between the cochlear and vestibular branches of the eighth nerve is also prominently shown in these researches. In the most severe cases there are only slight changes in the end filaments of the vestibular nerve, but never in the stem of the nerve or ganglion. This fact is interesting from the point of view of the neuron theory, because the nerve end apparatus of the vestibular branch never shows secondary involution processes analogous to those in Corti's organ due to neuritis of the cochlear nerve. Wittmaack maintains that he has previously proved that the cochlear nerve itself can recover, whereas the changes in Corti's

organ are permanent. Six plates are appended to the paper showing degenerative changes in the cochlear nerve and the secondary involution of Corti's organ. J. S. FRASER.

CLINICAL NEUROLOGY.

ON PARALYSES OF THE MEDIAN NERVE. (Ueber Störungen im Gebiete des Nervus Medianus.) WANDEL, *Deutsche. Ztschr. f. Nervenheilk.*, 1906, p. 436. (95)

IN this paper the writer records the features of 11 cases in which some type of injury had overtaken the median nerve. In four of these, fractures of the arm produced as their chief symptom complete paralysis of the thumb muscles (ape-hand) and diminished sensibility of the thumb and first three fingers. Five of the cases were of the nature of occupation neuroses, and these the writer states affect the median nerve, especially in cigarette-makers, ironers, carpenters, users of hammers, and carpet-beaters; in most of these there were loss of power in the forearm and hand, and sensory disturbances of a painful or anæsthetic nature. In the remaining two cases vaso-motor and trophic changes were the result of a median neuritis, and involved the skin of the thumb, index and middle fingers, and in one case the bones in the terminal phalanges of these two fingers. The cases are all described in full detail. JOHN D. COMRIE.

NEURITIS CAUSED BY CREOSOTE PHOSPHATE. (Neuritis verursacht durch Creosotum phosphoricum.) HUET, *Neurolog. Centralbl.*, Jan. 16, 1907. (96)

THIS paper records seven cases of neuritis of a peculiar type following the administration of phosphate of creosote for medicinal purposes, which occurred at Haarlem in the autumn of 1905. The amount taken varied from 25 to 120 grammes in about two weeks, and the symptoms set in about three weeks or more after the commencement.

The chief symptoms were—paralysis of the limbs, involving first the legs, later the hands, except in two cases, in which there was merely steppage gait; usually reaction of degeneration; loss of the Achilles-jerks, but retention of the knee-jerks, except in one case; loss of the plantar reflex; atrophy of the calf muscles; very slight muscle tenderness; cold feet, but few other paræsthesiæ; great difficulty in doing fine movements with the hands, such as knitting and buttoning.

Undoubtedly many people had taken the substance who did

not become affected by neuritis, and the writer thinks that some samples may have been impure. Some specimens were tested, but found to contain no free phosphorus nor arsenic. The writer discusses at length the possible rôle of phosphorus, creosote, and various impurities as the toxic agent, but does not arrive at any definite conclusion as to the substance actually responsible for the neuritis. JOHN D. COMRIE.

PRELIMINARY REPORT ON THE USE OF X-RAYS IN NEURITIS.

(97) CUMROE (of Penn.), *New York Med. Journ.*, Oct. 13, 1906.

IT has been more or less conclusively demonstrated by laboratory methods that the effects which X-ray exposure has upon the body may be classified as electrical, chemical, and inflammatory; chiefly the latter. The intima of the blood-vessels shows changes similar to those seen in endarteritis, and nutrition is proportionately lessened. Sensation in peripheral nerves is destroyed by electrical irritation. Prolonged exposure to X-rays produces a hyperæmia, acting as a counter-irritant.

Cumroe gives in detail the histories of six cases of neuritis, with the effect in each case of repeated X-ray exposures. His conclusions are: (*a*) the X-rays should be employed in neuritis only after all other judicious methods of treatment have failed; (*b*) the exposure should be made over wide areas, and frequently repeated; (*c*) the strength of the current and duration of exposure should be in direct ratio to the approximate distance of the affected nerve structures from the cutaneous surface overlying them: idiosyncrasies must be considered; (*d*) relief, if obtained, usually appears early in the treatment; (*e*) obstinate cases should be treated with a medium tube after apparent cure to assure permanency; (*f*) post-operative treatment in nerve resections is highly recommended. C. H. HOLMES.

TWO CASES OF TRAUMATIC AFFECTION OF THE CERVICAL CORD. (Zwei Fälle von traumatischer Halsmarkaffektion.)

(98) L. R. MÜLLER and R. LERCHENTHAL, *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxxi., S. 398.

1. DIRECT trauma to back of neck. Patient could just walk with assistance immediately after. One leg was completely paralysed at the end of twenty-four hours, the other not for two days. Severe pains occurred in both arms, and after four days in both legs as well. After presenting the usual train of symptoms,

bladder retention, respiratory paralysis, etc., the patient died twelve weeks after the accident.

The hypæsthesia did not ever become complete, contrasting with the motor phenomena. There was an area of hyperæsthesia from the second to the fourth rib, between the hypæsthesia of the eighth cervical above and the total hypæsthesia below. At the autopsy extensive pressure was found affecting the seventh segment; this was due to luxation of the sixth vertebra, which, being jammed against the fifth, greatly narrowed the canal. The grey matter of the cord had almost escaped, and the changes in the white matter, which were most marked in the posterior columns, were due rather to a sequential myelomalacia than to direct destruction. This accounted for the relatively late onset of the paralysis. The consecutive changes in the cord are described, the amount of ascending degeneration in the comma tract being noteworthy. The pains in the lower extremities were obviously not of root origin, and the author accounts for them by supposing an irritation in the leg fibres as they passed through the affected area. The upper level of the motor paralysis is fully described, and its bearing on localisation. The facts here confirmed the accepted teaching on the subject.

2. A powerfully-built man struck his parietal bone against a roof and fell unconscious. In the hospital, fifty minutes later, his state was as follows: Normal psychically. Pupils greatly contracted, but reacting to light. Arms strongly abducted and flexed at all joints (Thorburn's position). They were quite paralysed except for flexion of the elbow, and on the left side extension of the elbow and radial flexion of the wrist. Anæsthesia was both total and complete below the third rib and along the inner side of both arms, and all reflexes were abolished below this level. Marked priapism. No local signs in the neck could be detected. Death in thirty hours.

Post-mortem was found a fracture of both laminæ of the fifth vertebra; its spinous process, though movable, was kept in position by the well-developed muscles. Vertical fissure in the body of the sixth vertebra. No narrowing of the canal or flattening of the cord as in Case 1. No hæmatorhachis. The cord was normal microscopically down to the middle of the fifth segment. Below this to the seventh segment were numerous central hæmorrhages The axis-cylinders were swollen, but no changes were shown by Marchi's method.

As regards localisation, the case points to the elevators and external rotators of the shoulder joint (deltoid, spinati) being innervated from the fifth segment, the adductors and internal rotators (pectoralis major, latissimus dorsi, and subscapularis) from the sixth, the flexors of the forearm (biceps, brachialis anticus, and

coraco-brachialis) from the fifth, and the extensors from the sixth. The pupil changes were due to the centre in the seventh cervical and first dorsal segments being cut off.

Priapism in these cases has been attributed to either an excitation of the fibres concerned or to paralysis of the vasomotor nerves. The former is improbable, for it would be the sole evidence of excitation present in the midst of extensive paralysis. Bearing on its pathogeny are the facts that it is found only in high-seated lesions, and that it occurs (in the presence of bitches on heat) in dogs, the lower third of whose cord has been extirpated. The fibres connecting the brain with the pelvic plexus must thus leave the cord at about the mid-dorsal level and perhaps run with the splanchnics. ERNEST JONES.

SYRINGOMYELIA IN OLD AGE. (Die Syringomyelie im Senium.)

(99) E. FRIES, *Arb. a. d. neurolog. Instit. a. d. Wien. Universität*, Bd. 13, 1906, S. 170.

As vascular changes are believed to be an important factor in the causation of certain forms of syringomyelia, and as such forms would, therefore, be most apt to occur in conditions of weakened resistance and pathological changes in the vessel walls, *e.g.* in old age, Fries thinks that too little attention has been paid to the study of syringomyelia in old age. There are too few facts to go upon to enable one to say whether senile vascular changes stand in any relation to the actual origin of syringomyelia; but Fries gives full clinical and pathological notes of two cases occurring in advanced life, and, on the basis of these and of a few other recorded cases, he believes that the following conclusions are justified regarding tbe influence of these senile vascular conditions on the clinical course and pathology of syringomyelia already existing:—

1. Syringomyelia occurring in old age differs in no respect as regards clinical course from the disease as seen in earlier life.

2. The pathological anatomy of senile syringomyelia is to a certain degree characterised by these two points:—

(*a*) The glia-tissue, of which the gliosis consists, completely disappears, and is replaced by connective tissue, which arises from the adventitia of the vessels—*not* from the pia mater, as Sterling assumes.

(*b*) Lateral fissures formed by softening occur, and they need not stand in any relation to the central cavity. The area of softening may remain as such, or it may be replaced by a sclerosis, which again may later break down in consequence of the changes in the vessels.

Thus, senile vascular changes do determine in some degree the morbid changes found in advanced life, although the truly syringomyelic conditions—hydromyelia and gliosis spinalis—differ in no respect from the conditions found in earlier life, and are, in age as in youth, based on congenital defects of development.

A. W. MACKINTOSH.

POTT'S DISEASE AND SYRINGOMYELIA. (Mal de Pott et Syringo-
(100) **myélie.)** ALQUIER and LHERMITTE, *Rev. Neurol.*, Dec. 30, 1906, p. 1141.

CLINICALLY what is known as syringomyelic dissociation of sensation has frequently been remarked in Pott's disease. Cervical spinal caries in particular may present the picture of syringomyelia. Pathologically, in the former condition there may exist medullary cavities surrounded by a neuroglial wall, independent or not of the central canal of the cord. S. A. K. WILSON.

ON THE SYMPTOMATOLOGY OF THE EPICONUS MEDULLARIS.
(101) **(Zur Pathologie des Epiconus medullaris.)** MINOR, *Deutsche Ztschr. f. Nervenheilk.*, Bd. 30, p. 395.

THE author has described certain more or less well-defined symptoms occurring together in certain lesions of the lower part of the spinal cord, and has grouped them clinically. The combination of present knee-jerks, intact sphincters, and palsies or anæsthesias in the area of the sacral plexus depends on a lesion between the second and third sacral roots as a lower limit, and the fourth and fifth lumbar as an upper limit. To this section of the cord he proposes to apply the term epiconus medullaris. Several instances of a lesion of this region are given in detail. The roots which leave from the segments involved are included in the term. Thus a case of poliomyelitis, in which the knee-jerks are present, but the Achilles-jerks absent, the peronei and calf muscles wasted, the sphincters intact, and sensibility unimpaired, is a pure case of an epiconus affection. A steppage gait is characteristic of the condition. S. A. K. WILSON.

CONTRIBUTION TO OUR KNOWLEDGE OF TUMOURS OF
(102) **THE CENTRAL NERVOUS SYSTEM. (Ein Beitrag zur Lehre von den Geschwülsten des Zentralnervensystems.)** W. ROSENBLATH, *Deutsche Ztschr. f. Nervenheilk.*, Bd. xxxi., S. 335.

THIS article of sixty-two pages is a detailed description of nine cases which the author has selected from a series on account of the special features they show.

1. Male, aged 32. Headache, double optic neuritis, double exophthalmos more marked on the left side. At autopsy was found a tumour, the size of a hen's egg, at the pole of the left frontal lobe, and implicating the front of the caudate nucleus; microscopically it was shown to be a glioma. The orbits were normal.

There are nineteen cases on record of exophthalmos due to brain tumours. They were usually in the frontal lobe, but may be in any situation. The symptom has occurred with hydrocephaly and cerebral abscess. Flatau's explanation is that the cause is increased pressure occurring in individuals whose orbits are drained wholly by the cavernous sinus and not the facial vein.

2. Male, aged 53. Perfectly well till a fall on to his head five weeks before his death. Three weeks later, onset of numerous right-sided Jacksonian fits, in which the head and eyes were rotated to the right. Paresis of the right limbs, with no spasticity or changes in the reflexes. Discs normal. As lumbar puncture withdrew blood, late meningeal hæmorrhage was diagnosed and the patient was trephined. At the autopsy there was a large calcified tumour of the left frontal lobe towards its front. The Rolandic area was intact.

The case supports Müller's and Oppenheim's teaching that large frontal tumours can cause Jacksonian fits by pressure, and that a Rolandic lesion can be diagnosed only when the paresis affects progressively one limb after another.

3. Male, aged 43. Quite well till twenty days before death. Headache, double optic neuritis, mental symptoms, terminal coma. A small glioma was found in the gyrus rectus of the frontal lobe. Its recent development provided a good opportunity for the study of its structure. This is discussed in great detail. Ströbes' hypothesis as to the origin of glioma tissue is controverted.

4. Male, aged 56. Symptoms for two months before death. Paresis and clumsiness of the right arm. Paraphasia. There was a fairly large tumour in the left frontal lobe, pressing on the lower two frontal convolutions, the island of Reil and the first temporal convolution, and reaching to the lower end of the Rolandic fissure. It was a glioma with a peculiar plexiform structure. In spite of the destruction effected, the patient could speak spontaneously and understand all that was said to him. The author discusses the difficulty of reconciling the findings with the prevailing views on aphasia.

5. Male, aged 22. Symptoms for six months before death. Headache, vomiting, optic neuritis, progressive right hemiparesis, with no changes in the reflexes. Large tumour in left centrum ovale. It was a perithelioma that had undergone cystic degeneration. The relation of rapid formation of large cysts and the progress of certain symptoms is pointed out.

6. Male, aged 36. Headache, apathy, post-neuritic atrophy, no localising symptoms. Large tumour growing from the arachnoid and destroying a great part of the right temporal lobe. It was an endothelioma. No metastatic growths were found, as sometimes occur. The author examined the growth in vain for normoblasts, which have been recorded in such tumours, and have been adduced as evidence that they originate in the endothelium of blood-vessels.

7. Male, aged 43. Slight left hemiparesis, especially in the arm. Ataxy, asymboly, astereognosis, and tremor of the left arm. Papillitis developed late, with fixed pupils. The tumour was an endothelioma growing over the right parietal lobe and extending to the upper and back part of the frontal. It was therefore too large to be of any value in the localisation of the cause of the asymboly (Tastlähmung), though it supports Wernicke's view that this is cortical. The Argyll-Robertson pupil was inexplicable in the case.

8. Male, aged 49. Symptoms for only six weeks. Sudden onset of left brachial monoplegia, followed in a few days by paralysis of the leg also. This became complete, but sensation remained intact. A month later it was noticed that there was complete left-sided respiratory paralysis; not a sound could be heard in the chest on that side. The tumour was situated on the medulla, and affected the pyramidal tract just below its decussation. It grew from the pia between the cerebellum and medulla. In structure it was a spindle-cell sarcoma, the cells and tissues of which were impregnated with pigment. No primary melanotic growth was to be found. The respiratory paralysis is explained by the author as being due to pressure on the upper cervical cord of that side.

9. Male, aged 22. Headache, apathy, optic neuritis, spinal tenderness. Slight paralysis in the left arm, complete in both legs. Knee-jerks present. Plantar reflexes and left Achilles-jerk absent. Anæsthesia below the fourth rib. There was diffuse tumour infiltration of the whole length of the cord and extensively at the base of the skull, implicating both temporal lobes and the cerebellum. Microscopically it was an alveolar round-cell sarcoma, though its structure varied much in different localities.

ERNEST JONES.

THE FOCAL DIAGNOSIS OF OPERABLE TUMOURS OF THE CEREBRUM. (103) CHARLES K. MILLS, M.D., *Univ. of Penn. Med. Bull.*, April-May 1906.

THE scope of this paper is limited to a discussion of the focal diagnosis of operable tumours of the cerebrum. By operable, Dr Mills implies tumours situated so as to be accessible to the surgeon.

Further, this paper is in large part a summary of many previous publications which the author has made on cerebral localisation, and as such necessarily lacks much of the detail and evidence upon which his conclusions have been based. The author briefly describes the general symptoms of intracranial neoplasms, without entering at all fully into their localising value. He divides the brain into the frontal, parietal, occipital, and temporal regions, and for the purpose of diagnostic study subdivides these areas into the following six regions according to their physiological functions: (1) pre-frontal or higher psychic, (2) mid-frontal or intermediate psychic-motor, (3) post-frontal or motor, (4) parietal or sensori-stereognostic, (5) parieto-temporal or visuo-auditory, (6) occipital or visual. Before entering into the description of tumours of these various areas, he calls attention to the preponderating part played by the left hemisphere in the faculty of speech, and expresses the opinion that the cortical representation of cutaneous sensibility, muscular sense, and stereognosis has a higher evolution in the left hemisphere than in the right.

Pre-frontal or Higher Psychic Region is that part of the frontal lobes which lies anterior to a line drawn vertically to the mesial surface from a point half an inch in front of the anterior branch of the Sylvian fissure. Dr Mills recognises that much confusion has arisen regarding the functions of this region, owing to the misinterpretation of the mental symptoms due to intracranial tumour in any situation, and the failure to distinguish between those due to increased intracranial pressure and those resulting directly from involvement of the pre-frontal region. One regrets that with the recognition of this difficulty the author did not devote more space to its elucidation, in place of entering rather fully into indirect and distant symptoms due to lesions in other parts of the brain. The psychic changes characteristic of pre-frontal lesions are described as "evidences of higher psychic dissolution, which is shown in loss of memory and of powers of attention, abstract conception, comparison, judgment, and higher imagination." Beyond stating that tumours of the orbital surface of the frontal lobe give rise to psychic, olfactory, and optic tract symptoms, and that the psychic symptoms are probably more marked in lesions of the left pre-frontal region, there is no attempt to help one to localise a tumour of the right or left side. Exception must be taken to the statement that in order that any decided effect upon the mentality of the individual shall result, the lesion of the pre-frontal lobe must be destructive, and that characteristic mental changes do not occur without such destruction having taken place. This statement is too sweeping, and cannot be justified by clinical experience.

Mid-frontal or Intermediate Psychic-motor.—This area, which lies

behind the pre-frontal and in front of the ascending frontal convolution (post-frontal region), contains the graphic centre in the posterior part of the 2nd frontal convolution, the speech centre in the posterior part of the 3rd frontal convolution, and centres for the movements of the head and eyes intermediate between them. The symptoms of tumour of this region (on the left side) are temporary or permanent motor aphasia with Jacksonian attacks; Jacksonian epilepsy, affecting head, eyes, face, hand, and upper extremity; and some psychic changes. Destruction of the graphic centre alone results in inability to use the hand for graphic purposes, although full power of movement is retained. Tumours of Broca's convolution cause motor aphasia, sometimes recurring utterance, but no agraphia. Tumours growing from the membranes are characterised by an irregular development of agraphia or aphasia, and function is retained longer than in subcortical lesions. The importance of studying the initial and invasion symptoms is well brought out.

Post-frontal or Motor.—The localisation of such tumours is easy, the distinction between cortical and subcortical being based upon the frequent spasm and slighter spasticity in the former, and the rarer spasm and greater spasticity in the latter. The observations upon the conditions giving rise to Jacksonian epilepsy will be agreed to by all.

Parietal or Sensori-stereognostic.—This area is bounded in front by the fissure of Rolando, below by the fissure of Sylvius, and behind by a line drawn from the parieto-occipital fissure to the posterior end of the fissure of Sylvius. Cutaneous sensibility is represented in the post-central gyrus; muscular sensibility in the anterior portions of the superior and inferior parietal convolutions and stereognosis in the posterior part of the superior parietal. Cutaneous representation is apart from that of movement, and there are distinct areas for the sensory representation of the face, arm, leg, trunk, etc. Further, the author thinks that special areas of skin are definitely correlated with subdivisions of the sensorial and stereognostic cortex, and that the restricted centres and subcentres for sensation and stereognosis are correlated anatomically and functionally with special cortical motor centres.

Stereognosis is a conceptual process brought about by the recalling of memorial images obtained through such senses as contact, pain, temperature, location, vision, etc. There is no definition given of what is meant by muscular sensation or sensibility. The positive focal symptoms of a cortical parietal tumour are moderate impairment of cutaneous sensation, especially of pain and temperature sense, diminution of muscular sensibility, and stereognosis. Motor or visual affections occur if the growth

spreads respectively anteriorly or posteriorly. An absence of the plantar reflex and a diminution of the corresponding deep reflexes is referred to as characteristic of a strictly limited parietal tumour, but both these signs are met with in tumours situated elsewhere in the brain. In subcortical tumours the sensory symptoms are more complete. One wishes that more had been said about the paralytic negative and irritative positive symptoms of lesions of this area.

Region of the Junction of the Temporal, Parietal, and Occipital Lobes.—Tumours of this region on the left side affect the highest visuo-auditory centres, and word-blindness, word-deafness, reading, etc., hemianopsia or astereognosis if the tumour spreads forwards or backwards.

Middle Temporal and Inferior Temporal.—As the author leaves out of consideration the uncinate and hippocampal regions, and any reference to dreamy states, there is nothing special to guide one in localising a growth of this region.

Occipital or Visual Area.—A tumour on the external surface of the left occipital region causes visual aphasic symptoms, hemianopia, or perhaps astereognosis. If situated on the right hemisphere the aphasic symptoms are wanting.

The author concludes by some remarks on the sources of error in the focal diagnosis of brain tumour, and largely follows the lines of Collier's paper on the same subject. The paper finishes with a short and incomplete reference to the symptoms caused by deep-seated tumours. T. GRAINGER STEWART.

THE OCULAR SYMPTOMS OF TUMOUR OF THE CEREBRUM.
(104) G. E. DE SCHWEINITZ, *Univ. of Penn. Med. Bull.*, April-May 1906.

THE name of this article is misleading, as the author confines himself to describing the papillitis and retinal changes, and to a brief reference to visual field phenomena. Although no new points are brought out in the paper, the author is in general agreement with most of Paton's observations on the effect of operation on the optic neuritis. T. GRAINGER STEWART.

A TUMOUR OF THE OCCIPITAL LOBE OF THE BRAIN CURED
(105) **BY OPERATION. (Ein operativgeheilter Tumor des Occipitallappens des Gehirns.)** OPPENHEIM and KRAUSE, *Berl. klin. Wchnschr.*, Dec. 17, 1906.

THIS paper records the symptoms and removal, with singular success, of a tumour from the left occipital pole of the brain. The symptoms and signs presented were, in brief, headache, giddiness,

vomiting, double optic neuritis especially marked, and associated with retinal hæmorrhages on the right side; later, left-sided hemianopsia; and still later, difficulty in writing and reading, with right-sided hemiataxia, hemiparesis, and hemihypæsthesia. The various anti-syphilitic remedies having been tried without benefit, the left occipital region was trephined, and a large tumour found adherent to the dura mater, occupying the posterior extremity of the right occipital lobe, and extending down its mesial surface to the cuneus. This tumour was of the size and shape of a large hen's egg, shelled out easily, and proved to be a spindle-celled sarcoma. The adherent piece of dura mater was also removed. The wound healed well and a good recovery resulted. At first the patient had some visual hallucinations, seeing coloured patterns before the eyes, halos round objects, and an appearance of convexity or concavity in everything he looked at. A fortnight after the operation the optic neuritis had much subsided, and a week later the hemianopsia had largely disappeared. Within six months the patient was in the full activity of his business once again. Illustrations are given of the tumour and of the operation.

JOHN D. COMRIE.

REFLEX EPILEPSY IN EAR AND NOSE DISEASES. (Reflex (106) Epilepsie bei Ohren- und Nasenkrankungen.) HUGO FREY and ALFRED FUCHS, *Arb. a. d. neurolog. Instit. a. d. Wien. Universität*, 1906, Bd. xiii., S. 78.

THIS was an opening paper read at the Oto-Rhinological Section of the Lisbon Congress, 1906. The authors divide the Epilepsies into three groups—Jacksonian, Symptomatic (uræmia, Stokes-Adams, etc.), and General. Ninety-two cases were collected from the literature: 42 concerned ear affections, 16 being chronic suppuration, 41 concerned nose affections, 18 being chronic rhinitis with hypertrophied turbinates. The majority of these cases do not, however, teach us anything, because of the incompleteness of the description. The authors have examined 112 personal cases of epilepsy, and found ear, throat, or nose affections in 30; these were all treated locally. Sixteen had no other treatment, and four of these showed improvement of the epilepsy as a result. The cases were observed for a year only.

Mequius had shown that dogs suffering from repeated epileptiform attacks as a result of nasal infection with mites were cured by antiparasitic treatment. Brown Sequard's well-known guinea-pig experiments are also referred to.

The author's conclusions are: (1) In children suffering from epilepsy, or predisposed to epilepsy as a result of infantile cerebral disease, chronic poisoning, head trauma, etc., it is probable that

diseases and foreign bodies in the ear, nose, or throat may evoke epileptic attacks in the same reflex way as other peripheral stimulation. (2) In such cases local treatment may remove one of the exciting factors of the attacks. (3) It is not justifiable to speak of the epilepsy being cured thus, as only the exciting focus is removed, the predisposition remaining. (4) It is possible that the local affections cited may be more important excitants of epileptic attacks than peripheral excitation elsewhere. (5) Epileptics should be examined and treated from this point of view.

ERNEST JONES.

EPILEPTIC PETIT MAL, WITH PARAMNESIC AURA.

(107) **ILLUSIONS OF FALSE RECOLLECTION. (Crises de petit mal avec aure paramnésique ; illusion de fausse reconnaissance.)** SEGLAS, *Rev. Neurol.*, Jan. 15, 1907, p. 1.

THE patient was a man aged 28, who for four years had suffered from peculiar psychical troubles. At certain times he suddenly felt that he had been in exactly the same situation before. The environment, the people, their gestures and words, his own thoughts, all appeared to constitute a scene through which he had been before, though it was in reality quite new. Sometimes this feeling came over him as he was falling asleep; it was often called into being by emotion, or by intellectual or physical fatigue. He had also remarked the appearance of these paramnesic attacks if he came to a sudden halt after walking briskly. The duration of the sensation was merely a few seconds. At other times, however, it was the prelude of a more complex syndrome. No sooner had the feeling arisen, than he felt as though he were going to choke; his face became pale and his eyes fixed. Questions addressed to him were unheeded. Sometimes he remained still; sometimes he moved aimlessly about, muttering incoherently. After some minutes he usually recovered and continued what he had been doing previously, but he was always more or less completely ignorant of what had just taken place. The sensation was not infrequently associated with a feeling of anxiety or malaise.

It seems clear enough that, the false recollection apart, the other phenomena were of the nature of petit mal, and the sensation itself may be regarded as a sort of aura. The case resembles one published by Hughlings Jackson, where an epileptic suffered from fits, the aura of which was this sensation, and from petit mal with an identical aura, followed by complete amnesia both of the fit and of the aura.

S. A. K. WILSON.

ON FREQUENT PETIT MAL ATTACKS. (Über gehäufte kleine Anfälle.) KARL HEILBRONNER, *Deutsche Zeitschr. f. Nervenheilk.*, Bd. xxxi., S. 472.

(108)

THE author looks upon hysteria as a functional disease in the sense that we cannot expect ever to find anatomical changes underlying it, for its manifestations are throughout psychical; whereas epilepsy is a functional disease in the sense that though up to the present no characteristic changes are known, we may expect, with further perfection of technique, some day to discover them. The two diseases are thus theoretically separable, however much they may appear clinically to fade into each other. The great difficulty of diagnosing hysterical attacks from epileptic, so far from diminishing with our increased knowledge of the conditions, has considerably increased, chiefly owing to the recognition that more phenomena are possible in hysteria than was previously conceived. In fact at present the state of affairs is such that though in many cases we can say that a given attack is certainly hysterical in nature, it is hardly ever possible to say that any given attack is certainly epileptic. In other words, diagnosis of hysteria is always more sure than its exclusion. Especially is this so in the case of "minor attacks" due to hysteria, which may in all points be quite indistinguishable from minor epilepsy. Thus the diagnosis of minor epilepsy can only be made in the absence of other evidences of hysteria, and even then only tentatively as a rule. Although the above conclusions have been fully established, it is surprising how little known are the facts concerning hysteria on which they are based.

In this monograph of forty pages the author confines himself almost exclusively to the "nicht epileptischen Absencen" that Friedmann treated recently (see *Review of Neurology*, 1906, p. 575), and considered to be identical with Gélineau's narcolepsy; he discusses only those cases that occur in children. The varying symptomatology is given in detail, with numerous illustrative cases. Heilbronner agrees with Friedmann that most of the cases are not epileptic in nature, but is very sceptical about the attempt to establish narcolepsy as an entity quite distinct from other functional conditions. His main argument, which he repeatedly emphasises from various points of view, against the epileptic nature of the cases, is that they show no tendency, even after many years, to manifest intellectual decadence. He allows that there is a certain group of epileptics of whom also this is true, but outside this well-defined group he is unwilling to admit the epileptic nature of any attacks, repeated many times, that do not cause any psychical degeneration. He agrees fully with Binswanger that the epileptics who show this degeneration with the greatest certainty are those

occurring in childhood, and suffering from numerous abortive attacks, so that were the cases in question epileptic they would be just the cases to show mental changes. He does not lay the stress that Friedmann does on the loss of consciousness that occurs in petit mal, contrasting with the clouding that is found in narcolepsy; the variations in the depth of unconsciousness in petit mal are so great, as is the difficulty of estimating its depth in a short attack in a child, that, in our present knowledge, the point is of little value. The extraordinary number of attacks that may occur, even up to a couple of thousand in a day, and the sudden or rapid onset of the condition, are two points strongly favouring its functional origin. The condition may be simulated by several others, notably the attacks in juvenile G.P.I. and Jacksonian epilepsy. Its relation to reflex epilepsy is also discussed.

In conclusion, although Heilbronner maintains that these cases are true functional states and not of organic origin, which for him is the important problem to decide, he is less certain as to their exact position. He cannot accept narcolepsy as a separate entity for reasons indicated above, and because writers on the subject do not agree as to what are the important distinguishing features of what they term narcolepsy; he inclines towards including it under hysteria. The absence of other signs of hysteria does not exclude this diagnosis—a point on which Binswanger also insists—for monosymptomatic hysteria is a well-recognised condition, and especially frequent in children.

ERNEST JONES.

THE ÆTIOLOGY OF CONVULSIONS IN INFANCY AND CHILDHOOD. JAMES M'ILRAITH, *The Medical Chronicle*, Nov. and Dec. 1906 and Jan. 1907.

(109)

AT the close of his somewhat intricate and unduly detailed paper, founded on the study of 250 cases, the author arrives at the following conclusions, which I state in full:—

1. Convulsions are most frequent during the first year of life, and not very frequent after the third year.

2. The predisposing are more important than the exciting causes

3. The predisposing causes are "an inherited neurotic taint" and rickets.

4. Healthy children born of healthy parents rarely suffer from convulsions.

5. In children suffering from convulsions there is frequently a history of ill-health or disease in the parents, especially in the mother during pregnancy, and this acts by lowering the vitality of the child and rendering it more liable to disease.

6. The family history of children suffering from convulsions is bad. Frequently the brothers and sisters suffer, and the mortality in these families is high.

7. Only a small proportion of cases can be ascribed to organic disease of the brain.

8. Only a small proportion of cases of convulsions can be ascribed to injury at birth.

9. The most common cause of convulsions in the first two months of life is reflex irritation from the alimentary tract.

10. The great majority of convulsions are due to reflex causes.

11. Dentition is rarely a cause of convulsions, and only when some predisposing cause exists.

12. Gastro-intestinal disorders are the chief exciting causes of convulsions.

13. Convulsions are by no means as frequent as generally supposed at the onset of acute fevers, such as measles and pneumonia. They are more common at the onset of pneumonia than at the onset of measles. When they do occur there is usually some predisposing cause present.

14. In several of the cases of convulsions associated with whooping-cough a predisposing cause is present. Convulsions are by no means frequent in whooping-cough when no predisposing cause exists and no complications occur.

15. Convulsions in early life may be the first sign of epilepsy, or may give rise to that condition in later life, and are more likely to become epileptic when there is no obvious cause for the attack.

16. The fundamental cause of convulsions is some predisposing cause.

A. DINGWALL FORDYCE.

HYSTERIA IN CHILDHOOD. (Ueber Hysterie bei Kindern.) (110) RUDOLF FISCHL, *Prag. med. Woch.*, Nos. 51 and 52, 1906.

In his article on this subject the author states that the earliest age at which the condition is met with is towards the end of the second year of life, and then only rarely.

In the fourth and fifth years the disease is by no means rare, and increases in frequency from this time onwards till puberty. From puberty onwards, sex differences are noticeable in the frequency of the disease, it being considerably more common in girls than boys.

Its occurrence throughout childhood generally must, however, be considered relatively uncommon; and, while heredity and environment appear to play little part in its development, continuous school work is undoubtedly an important factor in its causation.

It has been customary to consider the great characteristic of the disease in childhood its mono-symptomatic nature. The correctness of this view the author questions. Common symptoms are the *tâche cérébrale* and increased tendon reflexes. The relation of the disease to "Spasmophilie" and their connection with the absorption of calcium salts is considered, and the author takes up the position that probably many cases of hysteria occurring in the later years of childhood owe their origin to a previous condition of "Spasmophilie."

In the experience of the author, the disease occurs in relatively simple and slight forms, and does not, as a rule, simulate any serious disease.

That it is, however, a disease *per se*, he insists upon, and as a disease it must be carefully treated. The commonest symptoms are cough, recurring vomiting with acetonuria, "school headache" —more especially during the study of German syntax!—unilateral blepharospasm, and joint neuralgia.

Early diagnosis of the condition is important, and the prognosis is relatively good.

In the treatment, suggestion, moral suasion, alteration of environment and conditions of life are the essential elements. In some cases it is necessary to have resort to various other methods —unpalatable or strong-smelling medicines, laryngoscopic examination, faradisation, and so forth.

A. DINGWALL FORDYCE.

AMBULATORY AUTOMATISM. (Automatisme ambulatoire.) PAUL (111) COURBON, *Ann. Méd.-Psych.*, Jan.-Feb. 1907, p. 22.

THIS is a full clinical record of five patients presenting the symptom of ambulatory automatism, in each of whom the condition was attributable to a different cause. In the first case the attacks were periodic, and their first appearance had been coincident with the cessation of malarial seizures, from which the patient had previously suffered. They were probably of a hysterical nature, although events occurring during an attack were remembered subsequently. The second was a good example of hysterical somnambulism, the incidents of the excursions leaving no trace in the memory. The patient was also subject to attacks of petit mal. In the third patient the attacks came on as a direct result of terrifying hallucinations caused by alcoholism, in marked contrast to the first two cases, where the expeditions were quite irrational and purposeless and seemed to be compelled by some mysterious and irresistible force. The fourth case, one of dementia præcox, illustrated an entirely different variety of the condition. The attacks were simply manifestations of the dementia, and had

no origin specially different from the other symptoms of the disease. The last patient was a soldier in whom the attacks took the form of repeated desertions. Although a man of inferior mental development, he could not be placed in the same class as the preceding patient, because his psychical condition showed no signs of progressive deterioration. He was regarded as being the subject of constitutional instability, and was not held fully responsible for his actions. HENRY J. DUNBAR.

STRIKING RESULTS OF EXAMINATION OF THE BLOOD IN NEURASTHENICS. (Ueber auffallende Resultate der Blutuntersuchung bei Nervösen.) GOETT, *Muench. med. Wchnschr.*, Nov. 20, 1906.

(112)

THE writer records the cases and results of blood examination in four cases of young men with marked neurasthenic symptoms, who all presented the peculiarity of a good hæmoglobin percentage with a great diminution of the red corpuscles. The figures were 82 with 3½ millions, 100 with 3½ millions, 90 with 2½ millions, and 95 with 2½ millions. The writer discusses the cause of this peculiarity, and gives a bibliography of articles bearing on the subject. JOHN D. COMRIE.

WHEN ARE TRAUMATIC NEUROSES CURABLE? (Wann sind Unfallneurosen heilbar?) DÖLLKEN, *Neurolog. Centralbl.*, Dec. 1, 1906, Jahrg. XXV., S. 1099.

(113)

THIS paper, which is based on a very extensive experience, was read before a meeting of the Mid-German Psychiatrists and Neurologists. The writer first discusses some of the etiological factors, operations during labour, injuries during games and trade pursuits. As there is no form of injury that will fatally induce neurosis, other factors must exist. Of these the author discredits heredity almost entirely; alcoholism is also unimportant. The prognosis and treatment of chronic neuroses incurred during trade pursuits are so special as almost to make this a disease apart. The writer has in the past eighteen years attended 13,000 workpeople, in four factories. He has demonstrated that when full wages are paid during the incapacitation of a workman, the acute traumatic neuroses, which are fairly common, hardly ever become chronic. On this, and on the skill of the doctor who first treats the case, argely depends the prognosis. It is almost impossible to give an accurate prognosis on the nature of the symptoms alone. The writer's conclusion on this point is strongly that both the existence

and the incurability of chronic neuroses are due to social conditions, which explains why medicine has failed so manifestly in their treatment. "Soziale Übelstände und ihre Folgen lassen sich nicht mit Medikamenten und Elektrisieren aus der Welt schaffen." The rest of the article is taken up with a discussion of these social conditions and their remedies. ERNEST JONES.

MIGRAINIC PSYCHOSES. A. GORDON (of Philadelphia), *Journ.* (114) *Am. Med. Ass.*, Jan. 5, 1907.

THE author reports twelve cases which he has seen during the past four years, in whom fairly typical psychoses were observed during migrainic attacks. In the majority of these cases the mental symptoms developed at the time when the headache had reached its climax and disappeared with the headache. The mental symptoms which he has found to be invariably present are confusion, mild stupor, hallucinations (mostly visual), unsystematised delusions, and delirium. Illusions of identity, incoherence of language and thought, and disturbance of orientation have been observed. He believes that a certain group of cases may be of an epileptic nature; the cause of the migrainic attacks and of the psychoses is commonly the same, *i.e.* auto-intoxication. Constipation, obesity, defective heredity are the most common antecedents.
C. H. HOLMES.

AUTO-SUGGESTION AND SEA-SICKNESS. (Influence de l'auto-
(115) **suggestion sur le mal de mer.)** H. ZBINDEN, *Archiv. de Psychol.*, Aug. 1906, p. 153.

A FRIEND of Dr Zbinden, with no previous sea experience, engaged himself as ship's surgeon on a voyage from Hamburg to Chili. The ship was due to sail during the night, and at two o'clock the surgeon was awakened by noises as of a ship on its course, and was seized with severe vomiting. In reality the ship was still in port, and the noises were due to the loading up of cargo. Shame at his impressionability cured the surgeon of the tendency to nausea, and he made the voyage without mishap. Following out the hint, Dr Zbinden made observations on himself and his fellow passengers during a rough passage of three days from Norway to England. By repeating to himself that he would not be sick, walking about (to a polka step), eating and smoking freely, and generally comporting himself like the old sea-dogs on board, he succeeded in being the only passenger unafflicted. Some experimental suggestions he made to fellow-passengers met with success. He remarks

that the mere fact of seeing somebody sick may have on other people a suggestive influence analogous to that of a nervous tic, which may contaminate a whole form at school. To guide check experiments, he sums up his attitude as follows:—

1. Sea-sickness is in all probability a neurosis.
2. It can be provoked or inhibited by auto-suggestion.
3. The fact of distracting the patient's attention from his woes assists the cure as in other neuroses.

J. ROY TANNAHILL.

PERIODIC TRANSITORY DISTURBANCES OF CONSCIOUSNESS

(116) **AFTER TRAUMA. (Ueber periodische transitorische Bewusstseinsstörungen, Dipsomanie, u.s.w., nach Trauma.)** ARTUR PELZ, *Monatsschr. f. Psychiat. u. Neurol.*, Jan. 1907, Bd. xxi., S. 53.

UNDER the title of "Mental Symptoms after Trauma" have been described the most diverse phenomena, so that it would appear almost as if any group of symptoms may thus originate. Attempts have been made to separate off precise syndromes that could be characteristic of traumatic neuroses. The present article is such an attempt.

Post-traumatic periodic disturbances of consciousness almost always develop on a basis of hysteria, neurasthenia, epilepsy, or alcoholism. The author critically reviews a number of published cases, and concludes that only one of them comes, not into this group, but into the section he now tries to establish. On the basis of three personal cases he fully describes, he maintains two principal theses: first, that there exists a group of periodic attacks of twilight states (Dämmerzustände) occurring after trauma, and apart from any basis such as those mentioned above; and secondly, that typical dipsomania, having no relation to epilepsy, may thus develop.

The first case, which is the most striking, runs shortly as follows: The patient, a man of 48, was run over by a train fourteen years ago, and had to have his right foot amputated in consequence. After this he suffered from many symptoms of psychosis, such as giddiness, headache, anæsthesia of the right cheek, etc. Four years ago his condition became worse, especially as regards the intolerance for alcohol that he had manifested since the accident. The following phenomena then occurred. For many months at a time he would be a steady, abstinent, regular workman, good-natured and peaceful, and a particularly fond husband and father. Then gradually would develop loss of sleep and appetite, great increase of sexual desire, marked excitability, restlessness, and

irritability. This phase would culminate in an irresistible impulse to drink, all his belongings would be sold, until after lying comatose in the gutter he would be brought home by the police. He then would tear his clothing, fling his children out of bed, threaten to strangle them and his wife, and violently accuse the latter of infidelity. After coming round he would spend a couple of days weeping, imploring his wife for forgiveness, vowing abstinence, etc. This would be followed by almost complete amnesia for the whole incident, and some months of perfect behaviour, during which time he would have a strong distaste for alcohol.

The later stage of the condition was thus certainly a dipsomania. Atypical features were the sexual excitability before the attack, which is unusual in dipsomania, though not unknown; the large quantity of alcohol consumed in the attack—the dipsomaniac is frequently satisfied without producing marked physical results of intoxication; and the jealousy delusions, which in true dipsomania follow the attack, and are not co-existent with an abrupt crisis, as in the present case. There was no hereditary taint and no history of previous heavy drinking, both significant features. The author next discusses the accepted view of Kraepelin, Binswanger, etc., that dipsomania is always a manifestation of epilepsy. He dissents from this opinion. In the above case there was not the slightest indication of epilepsy in the present or past condition of the patient. Traumatic epilepsy hardly ever occurs except after head injuries. Traumatic reflex epilepsy can likewise be excluded, as the scar gave rise to no symptoms. Wagner has published a similar case of dipsomania occurring after trauma.

In the three cases here described no attempt was made, by Freud's treatment or otherwise, to probe the disaggregated focus of mental content that must have existed, so that the value of the observations and conclusions drawn therefrom is relatively slight. ERNEST JONES.

A CASE OF TONIC CONTRACTIONS OF THE TRUNK. (**Zur** (117) **Kasuistik der tonischen Krämpfe des Rumpfes.**) FUCHS, *Wien. klin. Wchnschr.*, Nov. 29, 1906.

THE writer gives the details of the case of a woman, aged 35, who, apparently as the result of a fall upon the back during pregnancy, suffers from a condition of extreme lordosis (12½ cm.) with cervicodorsal kyphosis. The head also hangs forwards upon the chest, and walking is almost impossible. The condition is not always present, as she can at will straighten her back; but after a few seconds, whether she be standing or lying, the deformity gradually comes on. As it appears, the erector spinae stands out more

evidently. An X-ray photograph as well as gynæcological and surgical examination discovered nothing abnormal. The writer attributes the condition to a functional spasmodic contraction of the erectores spinae, and compares it to the condition frequently met with in the neck. JOHN D. COMRIE.

THE PROGNOSTIC SIGNIFICANCE OF THE ARGYLL-ROBERT-
(118) **SON PHENOMENON. (Zur prognostischen Bedeutung des Argyll-Robertson'schen Phänomens.)** ALEXANDER PILCZ, *Monatsschr. f. Psychiat. u. Neurol.*, Jan. 1907, Bd. xxi., S. 46.

IT is only ten years since Karplus dared to challenge the dogma that pupil fixation was diagnostic of epileptic as opposed to hysteric convulsive attacks. Since then its occurrence in the latter has been fully proved by Oppenheim, Westphal, etc. Marked aniscoria certainly may occur in neurasthenia, as for instance in eleven of Peligaeus' 320 cases; this also has been confirmed by Krafft-Ebing and many others. A feature of neurasthenic aniscoria is its variability, sometimes one pupil, sometimes the other being the larger.

The author now raises the question as to whether the Argyll-Robertson pupil is such certain evidence of organic disease as it is generally assumed to be. It undoubtedly is not pathognomonic of parasyphilis. Syphilis may give rise to it without there being any other affection of the nervous system manifest. The sign may occur also in the periodic psychoses, in paranoia, dementia præcox, etc. The literature on the subject is referred to extensively. Seven cases of psycho-neurosis are then described, in which various pupil changes, particularly sluggishness to light, were present. The cases were carefully observed for some years. Three of them the author selects as being particularly significant; in one of these the light reaction was quite lost, in the other two it was very sluggish. He does not definitely commit himself to the view that the phenomenon was due to the neurosis, but speculates as to this possibility. Speaking of the patients with psychical symptoms that are diagnosed as cases of general paralysis on the sole confirmatory evidence of the Argyll-Robertson sign, he urges that they should be looked at more sceptically from the point of view he indicates, and that lumbar puncture should invariably be performed. ERNEST JONES.

THE MASSETER MUSCLES IN HEMIPLEGIA. (L'état des muscles
(119) **masticateurs dans l'hémiplégie.)** MIRALLIÉ and GENDRON, *Rev. Neurol.*, Dec. 30, 1906, p. 1145.

IN fourteen cases of hemiplegia examined specially as to the condition of the muscles of mastication, it was found that the

temporals contracted equally while the masseters did not, or that lateral movements were defective, or that the inferior maxilla could not be pushed forward without its deviating to one side, or that diduction was impossible. The graver and the more recent the hemiplegia, the more noticeable were the defects.

S A. K. WILSON.

THE CONDITION OF CERTAIN REFLEXES IN SLEEP. (Ueber (120) das Verhalten einiger Reflexe im Schlaf.) KUTNER, *Deutsche med. Wchnschr.*, Jan. 17, 1907, p. 98.

KUTNER'S researches were made on male patients at the Breslau lunatic asylum. The knee- and ankle-jerks and plantar reflex were investigated. In all the cases the examination was repeated on the following morning, when the patients were awake, and, as far as possible, in the same position. The knee-jerks were examined in twelve cases, the ankle-jerks in twenty-two. The knee-jerks were present in all. In three cases the ankle-jerks were absent, but they were also absent in the waking condition, except when the patient was placed in a different position from that assumed in sleep. Kutner is therefore sceptical as to the diminution of the tendon-jerks alleged by some authors to occur in sleep. Such results he attributes to the difficulty of the technique. Only certain positions are available without waking the patient, and even in deep sleep complete relaxation of the joints, especially of the knees and ankles, is not always realised.

There was no difference between the results obtained in natural sleep and in that produced by such narcotics as veronal, trional, paraldehyde, and chloral hydrate. After the injection of scopolamine, on the other hand, Kutner found that the tendon-jerks were never diminished, but usually active, and sometimes exaggerated, the ankle-jerks more so than the knee-jerks. In eight cases well-marked ankle-clonus was present. In one case a violent patient with absent tendon-jerks was given a subcutaneous injection of $\frac{3}{4}$ milligramme of scopolamine. Ten minutes later the excitement was less, the tendon-jerks were active, and the plantar reflex, hitherto flexor, was now extensor. In sixty-three cases the plantar reflex was examined. In thirty-four cases sleep was spontaneous, in twenty-nine it was produced by veronal, trional, or chloral hydrate. In thirty-two patients, most of whom belonged to the dementia præcox group, the response was always flexor. Of seven patients suffering from general paralysis, four showed slow dorsal flexion of the great toes, two in natural sleep, and two after trional and chloral hydrate respectively. All when awake had active tendon-jerks. In the other three Babinski's sign was negative. The tendon-jerks in the waking condition were active in two and

absent in the third. Of the remaining twenty-four patients who suffered from delirium tremens, seventeen showed a positive, seven a negative Babinski's sign. Of the former, three were in a spontaneous sleep; in the remainder, sleep was produced by paraldehyde, veronal, or chloral hydrate. All when awake had active reflexes. J. D. ROLLESTON.

THE PLANTAR REFLEX AND BABINSKI'S SIGN. (Le réflexe (121) plantaire et le phénomène des orteils (signe de Babinski) du point de vue physiologique et physio-pathologique.) NOICA and SAKELARU, *Semaine Médicale*, Dec. 19, 1906.

THESE writers examined carefully the exact nature of the plantar reflex in a large number of cases, both normal and abnormal. In normal cases they find there are two distinct plantar reflexes, the internal plantar reflex and the external plantar reflex, associated respectively with the first sacral and with the last lumbar roots. The internal plantar reflex, obtained by gently stroking the inner side of the sole, consists of flexion and separation of the last four toes, with, if more pronounced, flexion of the great toe; and, if still more marked, bending and rotation of the foot inwards. The external plantar reflex, obtained by stroking the outer edge of the foot, consists of flexion and abduction of the little toe; and, if more pronounced, of flexion of the last two or three toes at their metatarso-phalangeal joint with extension at the other joints.

In a large number of patients, with various diseases of the nervous system, they found that the external plantar reflex was more readily elicited, and could be divided into eight different stages or degrees. Of these, the abduction of the smaller toes and the extension of the great toe, described by Babinski as indicative of an affection in the pyramidal tract, form two stages.

The external reflex is almost, without exception, more readily elicited by stroking the outer side of the foot, and frequently the writers found the Babinski sign by stroking the outer side, while the ordinary flexor plantar reflex was got by stroking the inner side of the sole.

The writers discuss the nervous and muscular mechanism by which the reflexes are produced, and give several references to the literature. JOHN D. COMRIE.

BABINSKI'S SIGN IN CHOREA. (Signe de Babinski dans la chorée (122) de Sydenham.) A. CHARPENTIER (Soc. de Neurol.), *Rev. Neurol.*, Dec. 30, 1906, p. 1176.

A BOY, aged twelve, had an attack of chorea following acute rheumatism. The irregular movements were generalised, but pre-

dominated on the right side. The tendon-jerks were equal on the two sides, but there was a definite extensor response of the toes of the left foot, while on the right side the plantar response was normal. This fact, which should be compared with the combined flexion of the trunk and thighs found by Babinski in certain cases of chorea, seems to indicate that a perturbation of the pyramidal tract exists in that disease. J. D. ROLLESTON.

THE PHYSIOLOGY OF LANGUAGE AND ITS RELATION TO
(123) **THE TREATMENT OF STAMMERING.** G. HUDSON-MAKUEN, *New York Med. Journ.*, Dec. 29, 1906, p. 1261.

THIS is a paper read before the Eastern Section of the American Laryngological, etc., Society. The author, after emphasising the neglect of defects of speech alike by qualified teachers and practitioners (with the natural result that such patients too often pass into the hands of quacks), holds that the study of these defects belongs more to the laryngologist than the neurologist. After alluding to the unfortunate confusion of nomenclature in connection with the subject, he adopts as the definition of stammering, "a more or less constant inability to speak freely owing to an inco-ordinate and spasmodic action of the respiratory, phonatory, or articulatory muscles."

He points out its prevalence amongst all nations, especially in childhood, and is evidently strongly impressed with the severity of the handicap placed by the condition on the patients, many of whom he even says "die early, because having a weak nervous organisation to begin with they are unable to endure the strain placed upon them."

He then discusses the importance of constructing an alphabet of sounds, and "receiving the suggestion from Melville Bell, Wyllie, and others," gives the table of sounds which he has drawn up, and calls (after Wyllie) The Physiological Alphabet. The table given in the article is practically identical with Wyllie's in every respect, even to the illustrative sentences; but although he holds that the use of his alphabet teaches the stammerer "the art of orderly thinking as well as orderly speaking," and puts great stress on the need for pursuing the "syllabication" method in the treatment of pupils, he makes no reference to the important physiological difference (as regards their proper method of enunciation by the stammerer) between the consonants classed as voiceless and voiced respectively. Evidently the main point in his view is the cultivation of syllabic enunciation, each syllable "requiring a separate and distinct respiratory impulse," which the patient is taught to give. HAMILTON G. LANGWILL.

TRUE TACTILE APHASIA. (**La vraie aphasie tactile.**) ERNEST (134) JONES, *Rev. Neurol.*, Jan. 15, 1907, p. 3.

A CASE of cerebral monoplegia was shown recently at the Neurological Society of Paris, where the patient was unable to recognise objects placed in the paretic hand, and Raymond, considering the defect as of central origin, called it "tactile aphasia." Déjérine, however, maintained that the peripheral disturbances were sufficient to make recognition of objects impossible. Claparède returned to the subject at a later date, and showed the condition to be really one of "tactile asymboly," arguing that we cannot speak of tactile aphasia unless the patient knows what the object is, and how to use it, but fails to name it from touch. Such a tactile aphasia must obviously be very improbable, since our memory of words is associated more intimately with auditory and visual images than with the sense of touch.

The author reports a case which he considers to be the first true case of tactile aphasia ever recorded. The patient was a man with hysterical right hemiplegia of traumatic origin, and of seven years' duration. He presented characteristic symptoms of analgesia, allocheiria, etc. During treatment he began to improve steadily, and in an interesting way. At first he had complete anæsthesia, although when his attention was diverted it was easy to demonstrate that he did respond to external stimuli. This was the phase of psychical anæsthesia. Later, he was able to feel objects, while he could not recognise their form or localisation. This corresponded to the "Tastlähmung" of Wernicke. Later, he recognised the form of objects, but had a certain amount of persisting faulty localisation, and slight objective paræsthesia. If a coin were put into his hand, he said it was a piece of metal, round and flat, but did not know what it was. This should be described as tactile asymboly, for there was clearly no astereognosis. Finally, the asymboly vanished, and the patient was able to recognise objects placed in his hand, and to use them accurately, but he could not thus name them. For an instance, a friend gave him a screw-driver of a rather peculiar shape, and while he employed it properly, he could not give it a name, although he was familiar with tools. The moment he heard the name mentioned, he knew it at once. The recognition of an object by touch depends first on the elementary sensations of touch, temperature, etc., then on the complex synthesis of these, and the resulting appreciation of form, surface, consistence, etc., then on the recognition of the essential nature of the object, and finally on the recollection of its name. Stereognosis and the sense of localisation are complex, and depend on a psychological synthesis, which naturally must be central. A peripheral lesion may abolish all

varieties of perception and recognition: if we cannot feel an object, we cannot recognise it. But it is possible to have a central lesion interfering with the identification of an object, though elementary sensations are not deranged.

Tactile aphasia seems to correspond to the optic aphasia of Freund, and it should be distinguished from tactile asymboly in the same way as visual asymboly ("Seelenblindheit") is to be distinguished from visual aphasia. S. A. K. WILSON.

PSYCHIATRY.

CONFABULATION. (Sur la confabulation et ses rapports avec la (125) **localisation spatiale des souvenirs.)** A. PICK, *Archiv. de Psychol.*, Aug. 1906, p. 141.

THE term "confabulation" is a neologism fathered by certain German psychiatrists to denote the attempts of patients suffering from amnesia to supplement the gaps in their memory by recollections which are purely fanciful. It originates in the unconscious necessity the patient experiences of projecting into the past an ordered register of collateral events in which he can localise the ideas evoked by the examining physician or by himself. The need on which confabulation is based is closely akin to the common feeling of discomfort accompanying failure to get definite hold of some elusive recollection. In the diseased person the easy and speedy resort to confabulation prevents discomfort from appearing or becoming intense, while the normal person has to possess his disturbed soul in patience till the lacuna is bridged, because he can do nothing to meet the emergency. Another explanation of the need for confabulation is set forth in Stout's "Groundwork of Psychology." This author refers the phenomenon to association, and shows that the events of a day, for example, however various in kind, leave in the memory a collective trace which can be reproduced *en bloc*. With this impression as a starting-point, component details can be made to surge up one by one in the memory. To localise a certain event in the past, as James points out, is to think of it in its immediate context. This remembrance by connection derives from the fact that every one of our actions, not only objectively but also psychologically, is for our ego but the fragment of a complicated whole. In states of weakened perceptivity the recognition of persons or objects is further enfeebled when they are placed in an unusual *milieu*. The everyday environment favours the linking up of associations. This localisation in space as opposed to that in time plays an important rôle in the functioning of the memory. How often are ungifted people un-

able to speak of a fact without recounting *ab ovo* the whole series of events of which it is a part.

The author analyses a case where a healthy workman of 58 was severely wounded in the right frontal region by a block of wood. Some bony splinters were removed, and a hernia cerebri treated. The patient remembered all about the accident, but was completely out of his reckoning in regard to space and time. As his condition improved, he occupied himself regularly with household tasks. The mechanical activities to which the patient had given himself over being deeply impressed on his nervous centres, were unaccompanied by confabulation, whereas a whole aureole of imaginary memories burst forth to furnish a setting of time and place.

Heilbronner explains the rarity of confabulation in eclamptics by the marked dissociation of ideas which prevents gaps in the memory being felt at all.

The relations between amnesia and confabulation fall into three groups: (1) simple defect of memory, (2) defect of memory with a struggle against confabulation, (3) defect of memory with confabulation more or less marked. These varieties cannot be supposed to be the consequence of arrested development, as in the case of the untruthfulness of abnormal children, and are more likely to correspond to a dissolution. J. ROY TANNAHILL.

APPARENT SUBCONSCIOUS FABRICATION. J. HYSLOP, *Journ.* (126) *of Abnorm. Psych.*, December 1906.

THE patient was a healthy, robust, middle-aged woman, of a very religious mind, and the wife of a clergyman. In early childhood, at the request of her father, she performed automatic writing on the planchette. Many years later experiments were carried on with the planchette, under the direction of her husband; she was unconscious of what was done by the planchette until it was told to her.

The subconscious writing showed supposed communications from deceased persons; accounts of life on the planet Mars, of which maps were drawn, zones indicated, inhabitants described, and evidence of the Martian language manifested. The author first saw the case in 1900; the Martian episode was resumed and a language was somewhat imperfectly developed. It is to be noted that in 1900 a somewhat unusual interest had been aroused by current literature in the planet Mars, which the patient may have read. Nevertheless more must be known about subliminal creations before one could establish an intelligible relationship between such coincidences.

Another personality called Harrison Clarke presented itself;

normal mirror and inverted writing could be used by the new personality. This personality, in response to the demand for proof of his identity, told a story, the falsity of which could be demonstrated; when the character was confronted with the facts he admitted their embarrassing character, but insisted upon "his reality as a discarnate spirit."

In looking for an explanation of these phenomena, if one turns to the subconscious mental action of the individual he will find a general source which cannot be doubted. To give a definite reason for the peculiarities of the phenomena in any given case such as this would be difficult. The process itself is one of "interrupted association in subconscious activity and its casual influence on the motor system." The nearest description to what occurs may perhaps be stated as "a partial suspense of muscular tonicity to allow the subliminal action to let occasional subconscious ideas slip through." C. H. HOLMES.

ON PSYCHOSES AFTER EYE OPERATIONS. (Über Psychosen nach Augenoperationen.) (127) M. LAPINSKY (of Kiev), *Allg. Ztschr. f. Psych.*, Bd. 63, H. 5.

THE patient was a poorly-nourished man, 60 years of age, with marked arterio-sclerosis, gastric disorder, and ulceration of the cornea, secondary to trachomatous contraction of the eyelid. During treatment for his eye trouble, which necessitated rest in a fairly dark room, he developed acutely a typical hallucinatory confusion such as one meets in arterio-sclerotics. One could exclude drugs as an ætiological factor. After discussing the case and reviewing the literature, the following conclusions are given: In psychoses following an operation on the eye there is, as a rule, no ætiological connection between the two. Psychoses after eye operations usually occur in advanced life; such psychoses have no specific features. The author considers that in these arterio-sclerotic cases the patients are especially susceptible to toxic influences; in the case recorded the toxic influence had its source in the disordered alimentary system of the patient.

C. MACFIE CAMPBELL.

CLINICAL CONTRIBUTION TO THE PUERPERAL PSYCHOSES. (Beitrag zur Klinik der Puerperalpsychosen, Generationpsychosen.) (128) G. HERZER (of Basle), *Allg. Ztschr. f. Psych.*, Bd. lxiii., H. 2.

AN analysis of the cases admitted to the Basle Clinic during the last twenty-five years, based on the classification of Kraepelin, with

special reference to the frequency and form of puerperal psychosis, their relation to the different periods of child-bearing, their course and prognosis and relation to heredity, number of births, and other ætiological factors, such as infection, trauma, etc. The material embraces 221 cases, forming 11·7% of the female admissions. The detailed results cannot be summarised. C. MACFIE CAMPBELL.

ON THE ÆTIOLOGY AND SYMPTOMATOLOGY OF CATATONIA

(129) **(Zur Aetiologie und Symptomatologie der Katatonie.)** PFISTER (of Freiburg), *Allg. Ztschr. f. Psych.*, Bd. lxiii., H. 2.

PATIENT was a young man of normal development until 1881, when, at the age of 15, he received a severe blow over the left parietal region, some fragments of bone having to be removed by operation. Patient appeared to recover completely; later a slight change in character was noticed—obstinacy, irritability, and later increasing intolerance of alcohol. There was also headache of variable intensity and tremor of the hands. In 1890 he was brought to the psychiatric clinic owing to the development of paranoic ideas, which consisted chiefly in ideas of persecution by his family; he was soon discharged, but in 1894 he was readmitted on similar grounds, with ideas of reference and auditory hallucinations; after five months he was again discharged improved, but was readmitted in 1897; he was suspicious and did not answer frankly, showed marked negativism, was evidently annoyed by constant auditory hallucinations. During the further course of his disorder, patient showed a well-marked catatonic picture with impulsive acts, stereotyped movements, mannerisms of speech, unmotived repetitions of the same phrases, negativism, mutism, and peculiarities of writing. He distorted the words by inserting after almost every vowel *s* or *h* or *i*, frequently doubled the diphthongs and the final consonants. His speech was defective, showing at an early date stammering and a difficulty resembling that of the general paralytic; the intonation was very monotonous, and he frequently repeated phrases. The dysarthria became more marked during the further course of the disease. The author considers that the trauma had given rise to a traumatic neurosis, which might be called neurasthenia, and that later on the basis of this, a slowly developing catatonic deterioration-process, had followed. Even when the whole disease was well established, the neurasthenic complex existed side by side with the other symptoms. There was tremor of hands and tongue, vasomotor excitability, increase of the reflexes, headache, and intolerance of alcohol. He considers that the trauma might be of ætiological importance in two ways, and takes for granted that auto-intoxication is at the basis of the

catatonic process. The trauma, therefore, might have caused either a general inability of the brain tissue to satisfactorily dispose of its waste products, or it might have caused an auto-intoxication by directly implicating hypothetical regulating centres of metabolism. He considers that as a rule the ætiological importance of trauma is exaggerated. In traumatic hysteria, neurasthenia, and the functional psychoses, trauma is merely a precipitating cause, the psychosis being equally due to the special constitution which gives the definite reaction.

With regard to the details of the case, he discusses especially the speech and writing defects. The writing defect had shown itself at an early date, and had at first been looked upon as a focal symptom; the further course of the disease made this view untenable, but the author considers that the local injury was perhaps a predisposing cause for the catatonic symptoms showing a certain elective severity in the speech realm. In addition to his intoxication hypothesis, he calls in the individual disposition as a possible explanation of the fact that in different catatonics different groups of symptoms are prominent. In some cases the greatest disorder is seen in the movements of expression, in others in their speech, in others in gestures and general behaviour. He has observed that those catatonics who show most marked speech disorder are those who are constitutionally "motor" in the sense of the French authors.

C. MACFIE CAMPBELL.

TRAUMA OF THE HEAD AS CAUSE OF INSANITY. C. BURR (130) (of Philadelphia), *Journ. Am. Med. Ass.*, January 5, 1907.

THE author asks two questions: (1) Is trauma in itself a true cause of insanity? He believes that trauma must have a suitable soil, such as unhealthy ancestry, cerebral arterial disease, syphilis, alcoholism, etc., otherwise insanity will not develop. (2) (*a*) Does any characteristic type of insanity follow trauma? (*b*) From the mental symptoms alone can it be told that a man is insane on account of having received a trauma? He believes that there is no characteristic type of post-traumatic insanity; the most common symptoms of this condition are confusion, delusions, simple dementia, and epileptic insanity. He has never seen "true melancholia" or paranoia follow an injury. He gives the case histories of seventeen patients, in whom the relation between the injury and the development of insanity was sufficiently close to make it reasonable to believe that the development of insanity was not a mere coincidence. Among these cases there were five in whom excessive alcoholism was thought to have played an important part; six were of advanced age; three suffered from general

paralysis; one showed distinct involvement of the left temporo-sphenoidal lobe and reacted to auditory hallucinations; one was constitutionally inferior; one became confused after an injury to the back. The author repeats that in none of these cases could traumatism alone be considered as the cause of insanity.

C. H. HOLMES.

TREATMENT.

REMARKS UPON THE SURGICAL ASPECTS OF OPERABLE (131) TUMOURS OF THE CEREBRUM. CHARLES H. FRAZIER, *Med. Bull. Univ. of Penn.*, April-May 1906.

MANY different opinions have been expressed as to the operability of cerebral tumours owing to the various interpretations which have been applied to the word operable. Walton and Paul, from statistics of the clinical and post-mortem records of 424 cases of cerebral tumour, classed 7 per cent. as operable, 80 per cent. as inoperable, and 13 per cent. as doubtful. Prof. Frazier points out that they used the term operable as practically synonymous with curable, and further, that their statistics were based upon cases in which the tumours had reached such a size as to cause death. It is obvious that from a study of such cases the number of operable cases is at a minimum. Prof. Frazier passes at once to the practical side of the question—the advisability of operation in cases of cerebral tumour. His opinion is that operation should be undertaken in every case in which relief from suffering can be secured for any considerable period. It is important to note that by operation he does not necessarily imply an attempt at the removal of the growth. In a few favourable cases operation may result in the complete removal of the tumour and the cure of the patient. In other cases the tumour may be removed in whole or in part without much prospect of cure, but with great temporary benefit to the patient. In other cases it may be possible to relieve the patient's suffering and to preserve his sight although it is impossible to remove the growth. Before advising an operation the first point to be determined is the situation of the growth and whether it is accessible to the surgeon, the most favourable situation being the surface of the brain on its extreme aspect. If the tumour be accessible, an operation should be performed to expose it. If the tumour be exposed, then the question as to its removal has to be decided. This depends on the size and nature of the growth. If the growth is infiltrating and of wide extent, the author holds strongly that its removal should not be attempted —(1) because such growths are very vascular and the risk of an immediate fatal issue from hæmorrhage is very great; and

(2) because such growths can never be removed *in toto* and are liable to recur, and grow more rapidly after they have been interfered with. These are the general lines which Prof. Frazier lays down.

In cases where the tumour has been localised, but is not exposed at the time of operation, he advises that small exploratory incisions should be made into the brain, as otherwise a tumour might quite well escape detection.

The technique of the operations and the various openings which he makes for exposing the different regions of the brain are fully described, and mention may be made of one or two of the points on which he lays stress. (1) The mapping out of the flap before the operation. (2) Osteoplastic operation. (3) The administration of ether as an anæsthetic instead of chloroform. (4) The frequent observation of the blood pressure as an accurate guide to the condition of the patient. (5) The performance of the operation in one stage. T. GRAINGER STEWART.

SOME NOTES ON THE DIAGNOSIS AND THE RESULT OF (132) **SURGICAL TREATMENT OF CEREBELLAR TUMOURS.**
B. SACHS, *Medical Record*, Dec. 22, 1906.

THIS article is devoted to the consideration of the surgical treatment of tumours of the cerebellum, or rather of cerebellar tumours, under which name the author apparently includes tumours situated outside the cerebellum.

The author desires to see the results of all operations on tumours of this region published, the failures as well as the successes, as he holds that an erroneous idea as to the success of operative interference has been created by the publication of successful cases only. During the past few years he has observed 22 cases of cerebellar tumour; of these, 7 were operated upon. In 4 cases attempts at removal of the tumours were undertaken, with fatal results; but in 2 of these cases, in which the side of the growth had been correctly diagnosed, the growths were not found at the time of operation. In 3 cases palliative operations were undertaken with benefit to the patients, pressure being relieved and vision preserved. In the other cases no operation was undertaken. These results have led the author to conclude that the present state of cerebellar surgery is not such as to warrant attempts being made at the removal of cerebellar tumours, and that palliative operation for the relief of pressure and of the neuritis is all that can be justifiably advised. The author admits that none of the cases were cured, and that those which were not operated on became blind; but holds that early palliative operation, although

not curing the patient, prolongs his life, preserves his sight, and rids him of his headache. There is, however, no attempt made to distinguish between the results of operation on cerebellar tumours proper, and tumours situated in the angle between the cerebellum and the pons, and indeed the author seems to class them together. The dangers and difficulties attending an operation for the removal of a tumour from the lateral lobe of the cerebellum are not to be compared with those incurred in an attempt to remove an extra-cerebellar tumour.

T. GRAINGER STEWART.

ON DIAGNOSTIC PUNCTURE OF THE BRAIN. (**Ueber die diagnostische Hirnpunktion.**) M. ASCOLI, *Berlin. klin. Wchnschr.*, Dec. 17, 1906.

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THE following method of performing this operation was devised by A. Kocher in 1899: After preparation of the parts in the usual way, a drill is inserted through the soft tissues, the bone perforated, and a Pravatz syringe introduced through the perforation. This form of exploratory puncture was employed by Th. Kocher in his Clinic, both as a therapeutic measure for the injection of tetanus antitoxin into the brain and evacuation of fluid from the ventricles, and as a means of diagnosis. The technique of the operation, as performed by Neisser and Pollack, is as follows: With a fine gimlet they pierce the soft tissues and bone, using instead of the drill an electric dental engine, and they have found the procedure of material service both from a diagnostic and therapeutic point of view. In spite of this, however, Ascoli says this important operation has not been extensively employed. With regard to the Neisser-Pollack method, he considers the current from the electric dental engine difficult to control, and has substituted in his cases an ordinary dental engine, to which the fine drill is attached. The foot motor is unsuitable with cocaine anæsthesia, as the operation is not so quickly accomplished. In using the ordinary dental engine the handle must be held in the same way as a pen, the elbow being firmly supported, so as to avoid injury to the dura or brain, and the ball of the little finger resting on the head of the patient. The drill should perforate the bone in a perpendicular direction. It is set in full motion, and lightly pressed on the selected spot. Sudden cessation of resistance indicates completion of the perforation, and the drill should be immediately stopped by pressure upon the knob of the handle, which is now removed, the drill being left *in situ*. Slight displacement of the soft tissues on removal of the drill sometimes renders it difficult to find the perforation and insert the exploratory needle, and Ascoli has

endeavoured to obtain fixation of the skin by means of a groove in the drill 8 mm. in depth, which on the completion of perforation sinks down and presses on the skin, but the results have not been altogether satisfactory. The method he now employs is to bind two parallel bands round the head of the patient at a distance of 1 centimetre, the spot chosen for puncture lying between them, and this, combined with fixation of the skin by the finger, appears to him to facilitate the finding of the perforation. In selecting the point of puncture, risk of injury to the vessels should be avoided (the art. meningea media and the venous sinuses), and one should be guided by the symptomatology and the anatomical characteristics. Neisser and Pollack employ Kocher's cyrtometer as a means of cranio-cerebral topography. As regards the depth to which the exploratory needle should penetrate, one must be guided by the symptoms in individual cases and the condition of the affected parts. When the focus of disease is deeply situated, radical surgical operation is rendered impossible, so that the advantage of deep puncture is more apparent than real, and it also involves the risk of injury to the vessels. Details of six cases treated by this method are given at the conclusion of the article. In one of these a diagnosis of cystic tumour of the right hemisphere of the cerebellum was made from microscopic examination of particles of the tumour removed by aspiration and the chemical characters of the fluid, and section showed a glio-sarcoma of the right hemisphere of the cerebellum. In another case puncture indicated disease of the left cerebellar hemisphere, and the case was diagnosed *by exclusion* as a tuberculous tumour, the sterility of cultures on the ordinary media negativing the presence of quickly growing abscess. In a further case the results of puncture were negative, while section revealed a tumour of the base of the brain, showing that a diagnosis based on negative results of puncture is not infallible. In another case the diagnosis of hæmorrhage of the arteria meningea media was confirmed on section. In one case penetration was accomplished in an unusually short time, the syringe being filled with cerebro-spinal fluid at a depth of 3 centimetres, and at the autopsy hydrocephalus was found, secondary to tumour at the base of the brain. Up to the present time, though the results of the procedure have not been wholly satisfactory, no unfortunate complications or results have occurred in the cases operated upon by the writer. He is of opinion that the operation should not be indiscriminately undertaken, but employed with care. On the other hand, he hopes that further experience will definitely establish the safety of the procedure, as in certain cases it is the one and only means of averting a fatal termination. DONALD ARMOUR.

Reviews

ZUR KENNTNIS DER VARIABILITÄT UND VERERBUNG AM ZENTRALNERVENSYSTEM DES MENSCHEN UND EINIGER SÄUGETIERE. J. P. KARPLUS. Pp. 162. Wien: F. Deuticke. (Otto Schulze & Co., Edinburgh.) 1907. 10 M.

THE anatomy of no other organ of the body has been so minutely studied as that of the brain, and owing to this fact, as well as to the complexity of its structure, there is no organ more suitable for the investigation of the hereditary transmission and the variation of characters. Karplus has already published an interesting monograph on the similarity of the fissuration of the fore-brain in different members of the same family, in which he showed that, in man at least, peculiarities in the brain sulci may be hereditary.

In the book under review the conclusions he then obtained are shortly summarised, and the results of the minute investigation on the variation in hereditary characteristics as revealed by the study of a much larger material is published for the first time. In twenty cases he was able to examine the brains of two members of the same family, in five cases the brains of three members, and finally in one case the brains of five members of a family. In addition to this he studied the nervous systems of four members of the families of an ape, of a bitch, and of a cat, and the brain of a goat and its twin kids.

His observations are in every case given in tables so arranged that the peculiarities common to, or the variations found in, the different members of a family can be easily compared. The conclusions of his previous work on the human brain are confirmed, viz. that there is a general similarity between the brains of different members of a family; but in the macaque monkey similarity is rarely observed between the furrow arrangement in the brains of a mother and of her offspring. In the monkey the two hemispheres of a brain are generally very much alike, while in the human brain there is almost invariably marked dissimilarity between the two hemispheres. This suggests that in the course of phylogenetic development the original symmetry in the fissuration of the two sides of the brain is gradually lost, and that the sulci of each hemisphere attain a greater permanency and become less liable to variation.

The book is an interesting contribution to the study of heredity and opens up a new and promising field for exploration. It is well illustrated.

GORDON HOLMES.

ÉLÉMENTS D'ÉLECTROTHÉRAPIE CLINIQUE. A. ZIMMERN. Paris: Masson & Son. (Otto Schulze & Co., Edinburgh.) 1906. 8 fr.

DR ZIMMERN'S book on Medical Electricity is well arranged, and contains a considerable amount of matter which one does not meet with in similar English treatises. Little attention is given to details of apparatus, but this enables more space to be allocated to discussing those physiological actions of electricity on which the methods of electrical treatment are necessarily founded, a long chapter being devoted to the excito-motor action of electricity, and a shorter one to the sedative applications of an electrical nature. Recent work is introduced on the theory of Ions and the introduction of medicines by electrolytic methods. Another chapter discusses modern researches on the resistance of the living body; whilst the final chapter, whose chief fault is that in some respects it is too brief, deals with the uses of electricity in various pathological conditions.

The book is interesting and valuable as reflecting the present state of electro-therapeutics in France, where, in many respects, it is better understood and more highly valued than it is in this country.

HARRY RAINY.

DIE RÖNTGENSTRAHLEN IM DIENSTE DER NEUROLOGIE. WILHELM FURNROHR. Berlin: S. Karger. 1906. M. 10.

DR FURNROHR'S book deals with a limited section of research in the department of X-ray work, but within this it is fairly complete. Like most German treatises it contains a lengthy literature index, compiled rather with a view to volume than value, in which the productions of the Fatherland occupy a predominant position.

The text discusses Diseases of the Brain and Skull, of the Spinal Cord, Vertebræ, and Peripheral Nerves, as well as the various Neuroses, and probably contains all that is of importance to know in these subjects in relation to X-ray work. The book is largely illustrated by actual X-ray photographs taken in the various conditions. The illustrations are of only moderate clearness, and do not in themselves give one any great impression of the value of radiography; but this is partly due to the inadequate methods adopted for their reproduction.

The general impression which one receives from a perusal of the book is that the contributions which are likely to be made by radiography to pure neurology are not of very great importance, although, in the case of tumours arising from neighbouring bony structures, this method may enable a diagnosis to be more certainly arrived at.

HARRY RAINY.

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AN INTERNATIONAL CONGRESS OF PSYCHIATRY, NEUROLOGY, PSYCHOLOGY, AND THE NURSING OF LUNATICS

will be held at Amsterdam on the 2nd to 7th September 1907, under the patronage of Her Most Gracious Majesty Queen Wilhelmina and of His Royal Highness Prince Hendrik of the Netherlands. The Congress will be divided into two parts: General Sessions and Meetings of Sections. In the former, chosen subjects will be dealt with by competent men who have been invited for that purpose; in the latter, questions will be discussed and criticism will be invited. Anyone taking an interest in Psychiatry, Neurology, Psychology, and the Nursing of Lunatics may become a member of the Congress. Those wishing to attend the Congress are requested to communicate with the General Secretary, Prinsengracht 717, Amsterdam.

Review
of
Neurology and Psychiatry

Original Articles

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

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DURING the past winter, and particularly within the last two and a half months, a number of cases of meningo-coccal cerebro-spinal meningitis have been admitted to the Sick Children's Hospital—5 under Dr Melville Dunlop, 7 under Dr Thomson, and 7 under myself. Four cases have also come under observation outside the hospital, making 23 in all. Post-mortem examinations have been made by Dr Stuart M'Donald on 14 of these cases, as well as on a number of others not seen during life. The following paper[1] is based on this material; the clinical notes refer chiefly to my own cases, though I have to thank my colleagues on the hospital staff both for their kindness in allowing me to examine patients under their care, and for permission to refer to them here. Within the same period 3 cases of pneumococcal meningitis have also been in the wards; these will be alluded to mainly for comparison.

Ages and Sexes of Patients, Dates of Onset, and Distribution.—The patients comprised 16 males and 10 females; their ages ranged from thirteen weeks to four years. Two of the pneumococcal cases were fatal; one is still alive, and apparently

[1] This paper forms part of a communication read at the Edinburgh Pathological Club on March 13, 1907, by Dr Stuart M'Donald and the author. The pathological aspects of the disease will be dealt with by Dr M'Donald in a future article.

improving slightly at the fifteenth day. Of the meningo-coccal cases 19 died, 4 remain alive. No patient has yet recovered completely. Twelve of the 19 fatal cases terminated in nine days or less ; 7 lived from periods of from eleven to forty-seven days ; in two-thirds of the patients, therefore, the course was acute, in one-third chronic. As the table shows, the tendency

Case	Age		Sex	Date of Onset	Duration
	Y	M			
1	3	6	M	Oct. 4	2 Days
(1a	—	10	M	„ 19	11 „)
2	2	10	M	Nov. 28	36 Hours
3	3	—	F	Dec. 14	Convalescent
4	4	—	F	Jan. 5	5 Days
5	1	6	F	„ 5	24 „
6	1	6	F	„ 6	Convalescent
7	4	—	F	„ 10	47 Days
8	—	8	F	„ 20	29 „
9	2	—	F	„ 22	82 Hours
10	2	3	M	„ 23	9 Days
11	—	3	M	„ 25	11 „
12	2	6	M	„ 27	17 „
13	—	10	F	„ 30	21 „
14	4	—	M	Feb. 9	56 Hours
15	—	9	M	„ 9	9 Days
(15a	—	9	F	„ 10	4 „)
16	2	10	M	„ 20	26 Days
17	3	6	M	„ 22	4 „
18	2	5	M	„ 25	5 „
(18a	—	7	F	„ 26	Still living)
19	4	—	M	„ 26	80 Hours
20	2	—	M	„ 27	Convalescent
21	3	—	M	„ 27	38 Hours
22	3	—	M	„ 27	5 Days
23	3	—	M	Mar. 4	—

a = Pneumococcal meningitis.

latterly has been towards the preponderance of the more acute type, for during February we had not so many protracted cases as in January. The cases have been scattered all over the city ; we have had cases from the old town, the southern suburbs, the Dalry and Gorgie district, Greenside and South Leith, and the

north side of the town. They have not been limited to the poorest localities. With one possible exception, to be mentioned later, no two cases have occurred in the same house or family. Even when cases have occurred in adjoining localities, a considerable interval of time has often elapsed between them. The parents of the affected children have followed a great diversity of occupations, and it has not been possible to trace any common source of infection in this quarter. There has been no apparent connection between the incidence of the disease and the mean temperature.

Clinical Features.—I do not propose to give the detailed analysis which has been made of the clinical features of these cases; but only to draw attention to what was made out as to (1) the antecedents; (2) the onset; and the leading symptoms, (3) head retraction and rigidity of the back; (4) the reflexes and other nervous symptoms; (5) lumbar puncture; (6) eruptions.

(1) *Antecedents.*—Most of the patients were in good health, and were well-nourished children. One had suffered from a sore throat the week before, and one had a definite history of a fall on the back of the head three days prior to his illness. There was no nasal or ear disease. No case occurred in an exclusively breast-fed infant. One of the pneumococcal cases had otorrhœa, another, an abscess in the arm, at the date of onset.

(2) *Onset.*—The outstanding feature was its suddenness. In many instances, the exact hour of invasion was known. To give an example: a little boy was out playing until 7 o'clock one evening, and came home to supper and went to bed as usual. At 3 A.M. he awoke delirious, with pains in the back and head, and died within fifty-six hours. Fever is invariable, and next in frequency comes vomiting, which may continue during the course of the disease. An initial convulsion occurred twice, and a history of headache was given by all but one of the patients over two. It was never severe, and was not often complained of by patients in hospital. Very often some slight cerebral symptom, such as a dazed, staring look, was spoken of. On the whole, except for its abruptness, the invasion of cerebro-spinal meningitis is not unlike that of any other acute infection.

(3) *Rigidity of the Back and Head Retraction.*—One or both

of these was present in every case at some period or another. Our previous experience of these was based on what we are accustomed to see in posterior basic meningitis, and from this point of view, neither (taking the cases as a whole) was as constant and striking a feature as was anticipated. Both are most marked in the protracted cases, in which they may become extreme, the patient boring the head into the pillow so that the occiput rests between the shoulder blades (Fig. 5 and 6). In acute cases they are often so slight as readily to be overlooked, especially in young infants (Fig. 1). There may be nothing more than a little resistance to movements of the head and neck, or the fact that attempts to do so cause crying. In two patients, one an acute, the other a chronic case, neither sign was present on admission on the first and the eleventh days respectively; in two of the most acute cases, which lasted for only thirty-eight and fifty-six hours, all that was ever present was, in the one, transient slight stiffness of the back, in the other, crying when the head was bent forward. In only three patients were stiffness and head retraction marked on admission. The earliest date at which definite permanent arching of the back and retraction of the head set in was the eighth day; the latest were the thirteenth and twenty-second days. In their classic form, then, the symptoms belong to the second week. Sometimes the head retraction preceded rigidity of the spine, sometimes *vice versâ*.

4. *Reflexes, etc.*—The *knee-jerks* were abolished in 3 cases, doubtful in 1, and active in 4. They were never distinctly exaggerated in the meningo-coccal cases, but in the pneumococcal cases they were, once with ankle clonus. The *plantar reflex* was absent thrice; in the others it was flexor. The most interesting feature concerning the reflexes was observed in connection with the *abdominal reflex*, which was abolished in all the meningo-coccal cases (see foot-note, p. 257). As a rule, this reflex can be elicited even in young children if the abdominal parietes are relaxed, and to the question of its absence we shall revert later. Kernig's sign (Fig. 4) was absent on admission in 6 cases, and in 3 of these it was never developed; in the rest it was fairly distinct. It may be quite obviously present even in young infants with flabby muscles. Thus it was noted as well

FIG. 1.—Case 8. Purulent cerebro-spinal fluid. Absence of head retraction at onset.

FIG. 2.—Case 8. Development of head retraction towards end of illness.

FIG. 3.—Case 20. Slight head retraction. Swelling of left knee-joint.

FIG. 4.—Case 7. Showing slight head retraction and Kernig's sign.

FIG. 5.—Case 13. Condition on day before death. Note opisthotonos, rigidity, and position of fingers and toes.

FIG. 6.—Case 13. Shows same points as Fig. 5.

FIG. 7.—Case 5. Position on day before death. Head retraction and flexor spasm of legs.

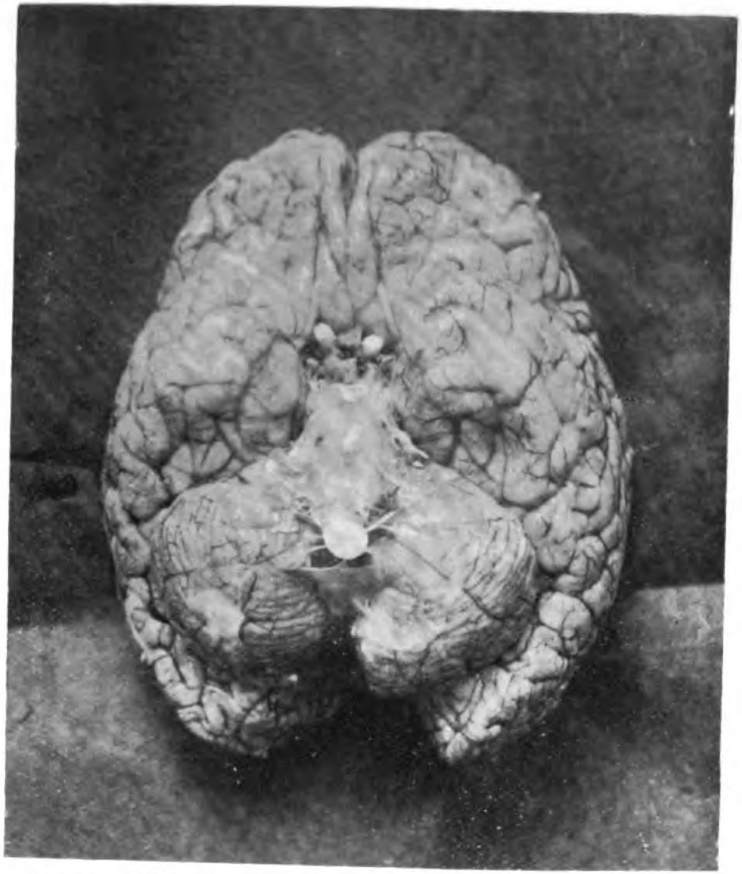

FIG. 8.—Brain from Case 7, showing patches of exudation over cerebellum and pons.

marked on the 3rd day in an infant aged 9 months, and it may occur from the onset.

Among other nervous symptoms, tremors and jerkings of the limbs, mostly of small amplitude, occurred in about half the patients. One child had a curious alternating hemispasm, in which first the left arm and leg began to jerk; after a quarter of a minute or so this ceased, and then the right arm and leg began. The cycle was repeated every 3 or 4 minutes for several hours. In other cases tremors were a terminal symptom. Paralyses of the limbs were absent. One infant was during her whole illness in a condition of general spastic rigidity—arms flexed, hands clenched, legs extended and adducted, toes turned in to soles. She presented a phenomenon the reverse of Kernig's sign, viz., when the leg was flexed at the knee it tended to spring back to the extended position as soon as the pressure was removed.

Mental Condition.—As a rule, patients are apathetic when unmolested; hyperaesthetic if disturbed. One was actively delirious, climbing about his cot whenever he was left alone. One could not but be struck by two circumstances: first, that considering the gravity of the malady, the patients expressed so little sign of pain; second, that consciousness was so well preserved, notwithstanding the presence of acute inflammation of the meninges. A boy of 4, whose illness lasted just two days, could sit up and answer questions perfectly clearly 18 hours before he died; a baby of a year smiled and played quite contentedly; a girl of 4 used to sing happily to herself. The two last were chronic, though fatal, cases.

5. *Lumbar Puncture.* — The meningo-coccus was always detected without much search in the cerebro-spinal fluid, although sometimes we failed, for no obvious reason, in first attempts to get fluid for examination. The organism was found throughout the whole course of the malady except in two instances, in which, after the 12th and 22nd days respectively, it could no longer be detected. The fluid always contained many polymorpho-nuclear leucocytes. While lumbar puncture is chiefly of importance as leading to a correct diagnosis, to which end it is, of course, essential, it is worth while inquiring whether the information derived from it (1) is of any value in estimating the probable duration of a case, (2) has any bearing on the clinical

features, and (3) whether the procedure is of any use therapeutically.

(1) *Lumbar Puncture and the Duration of the Illness.*—In the most acute cases, lasting only a few days, the fluid was milky or moderately turbid, and dropped out freely—never flowed from the cannula in a continuous stream. In some chronic cases it was much the same. The least turbid, almost purely serous fluid, which spurted out under high pressure, came from a chronic case. In the two most protracted cases the fluid was either purulent from the first, or became so later on; in both instances only a small quantity was obtained. The number of organisms found bore to some extent a relation to the acuteness of the case. In very acute cases they may be scanty, and in chronic ones they may be comparatively few. On the whole, in moderately acute cases they are most abundant. They were always found both in and outside the cells. In the most acute case observed (38 hours) very few were intra-cellular. Speaking generally, then, it may be said that turbid fluid under moderate pressure, swarming with organisms, betokens a bad case; few organisms, with severe constitutional symptoms, mean a very acute infection; while few organisms, with mild symptoms, mean a less severe attack. Serous or purulent fluid alike point to a protracted course.

(2) *Lumbar Puncture in Relation to Symptoms.*—We expected to find some sort of relation between the character, and particularly the pressure, of the cerebro-spinal fluid, and the presence or absence of such symptoms as Kernig's sign, spinal rigidity, etc. No such relation, however, seems to exist. We must recall the fact that intra-spinal pressure, as shown by the rate of outflow, is not an absolute index to what exists inside the cranium, because in chronic meningitis, matting at the base of the brain readily blocks the channels between the cranium and spinal canal, and may even lead to hydrocephalus without there being any rise in the intra-spinal tension. Here, however, we are concerned only with the pressure under which the cerebro-spinal fluid escaped on lumbar puncture. Of 2 cases in which there was no rise in pressure, one had rigidity of the spine and increase of the deep reflexes to a more marked degree than any other patient; the second had neither. Of 5 cases with turbid fluid under pressure, 2 had marked stiffness of the back, 3 very

little. In another case, in which the pressure, at first very high, fell steadily, head retraction and opisthotonos developed in almost inverse ratio. In that patient a good deal of matting was found post-mortem about the base. Pressure, therefore, as shown by the rate of outflow, does not appear to influence the symptoms much.

(3) As regards the *Therapeutic Effect of Lumbar Puncture*, the general impression gained from watching the cases was that it is small. In my own series of cases it was done about 40 times for diagnostic purposes and for treatment. One acute case, in which on 5 occasions within 36 hours about 6½ ozs. of fluid was withdrawn, was invariably relieved for the time being by tapping. In another case head retraction diminished considerably, but only for about 4 hours, after removal of cerebro-spinal fluid. Apart from these two instances, no benefit was obtained. On the other hand, two patients became temporarily collapsed after the withdrawal of small quantities. We were guided as to the amount drawn off by the rate of flow and the aspect of the patient; if there was no distress the cannula was left in position until the fluid ran less freely. In pneumococcal meningitis no good followed puncture.

Intra-spinal injections of 1 per cent. lysol and of collargol were tried without avail. The former had no apparent effect one way or another. The latter gave rise to unpleasant symptoms, such as tremor and violent delirium, on several occasions. More important, however, from a practical point of view, is the fact that on post-mortem examination of a patient who was under treatment with collargol, indisputable traces of the drug, which has a dark-brown colour, were found in the exudate at the base of the brain. It is therefore reasonable to suppose that injection of a suitable antiseptic might be of value in treatment.

6. *Rashes.*—Two forms of rash occur: herpes and purpura or erythema. The characters of the latter vary greatly. In 4 of the 8 cases there was no eruption whatever. Four, at least, of our hospital cases—3 chronic and 1 acute—have had well-marked labial herpes as an initial symptom; there has been no instance of its occurrence in any other region. A true hæmorrhagic rash was present in two cases in hospital and one outside—all of them acute. In one the whole body was covered with

purpuric spots, varying in size from a pea to a threepenny piece, with a large extravasation over the knee-joint. In a second case, on the morning of admission (second day), the rash consisted of reddish-purple macules, sparsely scattered over the chest and arms, not unlike commencing measles or the earliest appearance of chickenpox. In the evening the rash had become much more profuse, but still retained its erythematous character. Next morning most of the spots had faded; a few had become hæmorrhagic, and persisted till death. In several patients evanescent mottling, chiefly about the extensor aspects of the arms, was noted. This seemed to be connected with a tendency towards cyanosis which exists in cerebro-spinal meningitis. The patients were often slightly dusky on admission, and became very livid towards the end. The impression it gave was that it was due as much to toxæmia as to malaëration. In most cases the blood was examined. There was always a leucocytosis (14,000 to 38,000), and usually a positive glycogen reaction. In the case with serous exudation, the latter was absent.

Relation of Epidemic Cerebro-Spinal to Posterior Basic Meningitis.—Simple basal meningitis is a well-known, not very uncommon disease of children. Fifty per cent. of the patients are under 6 months, 84 per cent. under a year, and 90 per cent. under 2 years. It is therefore a malady which almost solely affects young infants. It runs a chronic course of seven weeks on the average, and is not always fatal. Dr G. F. Still has described a diplococcus, almost identical with the *diplococcus intracellularis meningitidis*, which has since been constantly found in the cerebro-spinal fluid in the disease, and most authorities now regard posterior basic simply as a sporadic form of epidemic cerebro-spinal meningitis. We were, therefore, especially interested in comparing the cases we have lately seen with those to which we were formerly accustomed. The acute disease is, of course, totally different clinically. Chronic cases, however, are so very similar in many respects, that it is impracticable to draw any hard and fast line between the two conditions. At the same time, there have been minor peculiarities. Taking the chronic cases as a whole, it may be said that almost any one of them, had it occurred alone, would have been looked on as a rather aberrant type of posterior basic meningitis. For one thing, many of the patients were of an age at which posterior basic meningitis

is rare. Again, meningo-cocci were, on the whole, more abundant in the cerebro-spinal fluid, and there were more intracellular forms. A notable point of difference was the existence of labial herpes in three or four cases. According to Barlow, this is very rare in posterior basic meningitis—indeed, he comments specially on the fact as remarkable, considering the close relation of the disease to the epidemic form. There is no doubt that posterior basic is a sporadic variety of epidemic meningitis, but it seems as though, when an epidemic prevails, the organism undergoes modification, so that it no longer tends specially to affect children under a year, or to reproduce exactly the same symptom-group which it does in common sporadic cases.

Relation of Cerebro-spinal Meningitis to Acute Abdominal Infection.—The suggestion that epidemic cerebro-spinal meningitis may be related to acute abdominal infection originated primarily from certain pathological observations made some months ago by Dr Stuart M'Donald, to whom the credit of priority is therefore due. For the sake of clearness, however, the pathological as well as the clinical facts which appear significant are stated here; the former will be elaborated by Dr M'Donald in a future communication.

The commonly accepted etiology of cerebro-spinal meningitis is that the organism gains access to the brain from the nasal cavity. Without denying that this may occur, we can only say that we found neither clinical nor pathological evidence supporting it. There seem, however, to be certain facts tending to show that in cerebro-spinal meningitis the cord lesion is primary, and that the diplococcus invades the nervous system through the spinal meninges.

1. In most cases the post-mortem appearances show that the cord lesion is of older standing than that of the brain.

2. The constant early and complete abolition of the abdominal reflex, already referred to, seems to point to early implication of the lower dorsal cord.[1] It is interesting to compare the pneumococcal cases (in which the lesion is primarily cerebral) in this particular. In one of them the reflex was active. Lumbar

[1] Since this paper was written, I have observed the abolition of the reflex in several other cases, and its return in three cases during convalescence. In one acute case I have also found it to be present, but the general rule that the reflex is absent, does not seem to be invalidated by this single exception.

puncture, a few days before death, yielded clear fluid, with no cells or organisms. In the second and third cases the fluid was turbid, and swarmed with pneumococci; in them the reflex was absent. It is therefore justifiable to conclude that the abolition of the reflex points to spinal lesion. The withdrawal of clear sterile fluid from a case of pneumococcal meningitis is noteworthy. Osler states that a negative finding of this kind is common in such cases, so long as the meningitis remains limited to the cranium. If this be so, the fact that meningo-cocci can be detected in the fluid in cerebro-spinal meningitis from the very onset is an additional argument in favour of the spinal origin of the infection.

3. Another small fact, tending, however, in the same direction, is the slight amount of disturbance of cerebral function in some of these cases. The retention of consciousness late in the disease has been mentioned; a child may be able to sit up quite contentedly days after pus has been removed from the spinal canal. (Fig. 1.)

If, therefore, as there seems some reason to suppose, the brunt of the infection first falls on the cord, the most natural channel of entry is from the abdominal cavity. The information bearing on this is for the most part pathological; but two pieces of clinical evidence may be given.

4. A possible exception to the statement that no two cases occurred in one family, has been spoken of. A child died of acute cerebro-spinal meningitis on a Friday morning after 2 days' illness. On Sunday an infant sister, aged 3 weeks, died after 12 hours' illness. She was seen by the same student who had recognised the nature of the first case, and was supposed merely to have some digestive disorder. Post-mortem, the brain and cord seemed healthy; the appearances in the abdominal cavity were similar to what are seen in many cases of cerebro-spinal meningitis, and the diplococcus meningitidis was found in smears made from the base of the brain.

5. No case of meningo-coccal meningitis in an exclusively breast-fed infant has come under our notice. The possibility of bowel infection is therefore evident. In the case just referred to the infant was nursed until the elder child took ill, when the mother's milk failed. During the last four days of life, therefore,

and for three days preceding her illness, she, also, was fed artificially.

Finally, I may again emphasise the fact that lumbar puncture is of paramount importance in regard to diagnosis. In very acute cases, in children at least, the characteristic symptoms may be so slight at the onset, that even after some little experience of the disease, one remains in doubt until the cerebro-spinal fluid has been seen. The procedure is safe and easy to carry out, and the withdrawal of a little turbid fluid suffices practically to clear up the nature of the case.

A PLEA FOR THE SCIENTIFIC TREATMENT OF STAMMERING.

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THE condition of stammering, though not exactly common, occurs amongst all classes with a frequency that deserves, I venture to think, a more adequate recognition from the medical profession than it generally receives. That the fault lies entirely with medical men I do not, of course, maintain; for few things are more striking than the acquiescence of the parents or relatives of a stammerer in regarding his malady as only "a bad habit," or "largely the result of nervousness"—a matter, therefore, in their opinion, on which *medical* advice need not be sought. Now, this fact (and it will be generally admitted, I think, that a doctor is seldom consulted upon the subject of stammering) is of much interest. For every general practitioner knows well the eagerness with which his advice is sought (and his "operative" assistance often urged) by an anxious mother who imagines her infant (of a few weeks) to be "tongue-tied"; and yet when, in later years, one (or even more) of the children in a family shows even marked stammering, the family practitioner is rarely consulted on the matter. Relatives and friends, more or less sympathetic but probably all equally ignorant of the subject, may be spoken to about the boy's infirmity, or the advice even of the clergyman may

be sought, but the family doctor in such a case is too often looked upon as a *dernier ressort.*

Or, again, should one of her children show signs of "limping" or "halting" in his *gait*, no time is lost in seeking the advice of the medical attendant, who is closely questioned as to the possibility of any incipient joint disease; and yet another little sufferer—perhaps partly because *his* pain is *mental* rather than physical, and so does not betray its presence by fretful crying—is frequently allowed to go on for years "limping" in his *speech*, developing an increasing "impediment" which may cripple his whole after-life. At the door of the stammerer's relatives, then, a goodly proportion of the blame for the neglect of this serious affection must undoubtedly be laid.

But, on the other hand, does it not too often happen that, should the advice of a doctor be sought regarding a stammerer, he is rather at a loss as to how the condition should be treated. And yet the fault is not mainly his, for the subject was very probably not dealt with at all in his undergraduate teaching, and to his usual text-books he will turn almost in vain; for although in some of the larger cyclopædias or "systems" of medicine of recent years the question is considered, the affection is scarcely mentioned in the ordinary text-books. Hence it may not be altogether without reason that, in dealing with such an affection, it has been customary on the part of the public to reckon the family doctor's advice as only secondary in importance to that of the boy's school-teacher or clergyman, who are both regarded as more experienced in the management of the "unruly member." Lack of practical knowledge of the subject, then, on the part of the medical attendant, as well as indifference of the patient's relatives, probably account for the comparative neglect of the treatment of this important and too common affection.

The result of this ignoring, to a large extent, of stammering by the medical profession, is only too clearly shown by the frequency with which one notices in the press advertisements of so-called "cures" for stammering. There is evidently a demand for treatment; is it satisfactory or creditable that our profession should concern itself so little with the proper scientific treatment of the defects of such a manifestly physiological act as speaking?

Now it is worth noting that, while the methods of some of those so-called "stammer doctors" pertain largely to pure quackery, it is undoubtedly true that others have really given some attention to the study of the subject, and by the adoption of certain modes of treatment (not by any means scientific) have produced sometimes results which are, on the whole, fairly satisfactory. Such being the case, one might expect that accurate scientific investigation of the subject, founded on physiological data, should give still better results. The treatment of a neurosis (such as stammering undoubtedly is) ought not to be considered as outside the domain of the medical profession. No small part of the curriculum of the student of medicine is taken up with instruction—systematic and clinical—in nervous ailments, organic and functional. Why should the study of stammering be so neglected? I have heard it stated that a medical man might be apt to be regarded as dabbling in quackery should he become known as qualified to treat cases of stammering. Can this be another explanation of the comparative neglect of the subject by neurologists and medical teachers generally? There can, I think, be little doubt that the ignorance of the *practitioner* on the treatment of this too common affection must really be laid at the door of his *teachers*.

Surely the family medical attendant ought to be as competent to give advice as to the rectification of faulty *speaking* as he is in the correction of faulty *walking*, *e.g.* in incipient "flat-foot"—an instance where accurate study of the anatomy of the foot revealed clearly the muscular exercises which are likely to strengthen and develop the faulty supports of the plantar arch. Now, undoubtedly "flat-foot" is a condition which comes under the notice of medical men much more than stammering, but if the seriousness of a malady is to be measured, not by the frequency of its occurrence, but by the amount of suffering it entails, I venture to maintain that in the stammerer we have a patient deserving of our utmost consideration. Probably few, except those who have been its victims, can realise the amount of mental suffering endured by many stammerers—not merely the fear of ridicule in boyhood, but the attendant nervousness and actual difficulty of enunciation of many school lessons, *e.g.* many Greek verbs

with their reduplicated voiceless consonants (*e.g.* πιπτω, πεπτωκα). The nervousness which is so often assigned by the patient's friends as a *cause* of his affection is much more truly one of its *results*; another of which, in many instances, is a morbid shyness and avoidance of company on the stammerer's part.

Granting, then, the importance of the subject of stammering and the need for its more systematic treatment by the medical profession, how can this be undertaken? I desire to draw attention to the method of treatment advocated by Prof. Wyllie of Edinburgh University, in his articles which originally appeared (in 1891) in the *Edinburgh Medical Journal*, and were subsequently published in his work on "The Disorders of Speech." As one of his earliest pupils when he was working at the subject, I have long since proved the great value of his teaching, not only by my own personal experience, but also by putting his methods into practice on others. I have previously endeavoured[1] to enlist a wider interest on the part of the profession in Wyllie's method, and drawn attention to its comparative simplicity; for the value of his admirable work is, to a considerable extent, I think, lessened by the fact of its being contained in a work on a special subject which rarely finds a place on the shelves of the general practitioner. Now, his method of treating stammering is essentially designed *not* merely for the specialist, but for the practitioner, who can carry out the treatment of the condition satisfactorily in the most outlying country village, *provided*—and this is the crux of the whole situation—the patient will *perseveringly* carry out the instructions given. Stammering is one of those conditions to the treatment of which the old adage is especially applicable about taking "a horse to the trough," etc. It is the *patient* who must carry out persistently (under medical supervision) the instructions laid down for him, and it is on his own perseverance that the rapidity and completeness of his cure will depend.

Those who desire a fuller exposition of Wyllie's views, not merely on stammering but also developmental affections of speech, will find them in his book ("The Disorders of Speech"); my endeavour here will be to give a résumé of his treatment of stammering, with some additional notes on certain points.

[1] *Brit. Med. Journal*, July 20, 1901; *Practitioner*, January 1903.

Symptoms. — No detailed account of the symptoms of stammering is required, but there are, of course, many varieties of the condition; these, however, fall into two main groups: (*a*) those who "stutter," *i.e.* show a tendency to the rapidly recurring enunciation of the particular letter-sound which has caused the difficulty in speech; (*b*) those who (in Wyllie's phrase) "silently stick" at the difficult letter. The first variety (who say, *e.g.* "p-p-poor B-B-Bob") is found more commonly upon the dramatic stage than in real life. The patient with the other and commoner variety tends (as Wyllie says) to manufacture a "great big capital" P (in saying "Poor Bob"), instead of several little "p's," the process of the evolvement being generally *silent* (hence the unsuitability of this type for representation on the stage). But though there is practically nothing *audible* to the bystander during the interruption of the patient's utterance, very frequently other evidences of the stammerer's distress and difficulty can be *seen* (congestion of the face, grimaces, etc.), which are well described by Kussmaul. These involuntary facial and other movements should be distinguished from certain *purposive* actions (*e.g.* tapping with the foot, swaying of the body, etc.) sometimes made use of by patients who have found from personal experience, or having been taught by a "stammer-curer," this muscular outlet helpful in overcoming their difficulties. It is, of course, well known that it is especially *initial* letters that cause trouble to stammerers; that consonants are far more frequently a source of difficulty than vowels; and also it is where using the ordinary conversational tone that stammering is apt to be worst. The condition is much more common in the male sex at all ages. Most important of all is it to note that scarcely ever does a patient stammer in *singing*.

Pathology.—This requires only the briefest notice. Stammering is essentially a functional affection—a neurosis the result of defective *working* of the complex co-ordinated mechanism concerned in the production of speech, and not a defect of *structure*. Pathological lesions, therefore, connected with it cannot be looked for, and herein lies one of the great inducements to the adoption of proper scientific treatment of the condition.

Treatment.—In each of the two classes of stammerer men-

tioned above, the difficulty of enunciation will be found to be essentially due to a *want of the necessary harmony between the functions of vocalisation and articulation.* The first essential, then, in commencing treatment of any case is to explain to the pupil briefly the process of *normal* speech utterance. It is not necessary to take him at any length into the physiology of the subject, but he must be made to grasp clearly the fact that during the act of speaking *three* separate mechanisms are harmoniously at work: (1) a current of air is being expelled from the lungs; (2) sounds are produced in the larynx (*vocalisation*) by the vibration of the vocal cords; (3) these initially laryngeal sounds (and others also, as will be seen later) may be modified by the actions of the lips, tongue, etc. (*articulation*).

The great value of the first of these co-ordinating mechanisms will be alluded to subsequently; what the stammerer has to grasp is that *his* difficulty is essentially due to *defective co-operation between vocalisation and articulation*, and in by far the largest number of instances it will be found that it is his *vocalisation* which is at fault—being deficient in *amount* as well as in *promptitude.* The simile which Wyllie makes use of to illustrate this appeals at once to any pupil. He compares the co-ordinated mechanism at work in speaking to the performance of a violinist: just as the player's right hand ("bow hand") produces sounds which are modified into notes by the left ("string hand"), so (in speaking) vocalisation corresponds to the "bow hand" and articulation to the "string hand." Now, we have seen that it is the stammerer's *vocalisation* which is faulty in the greatest number of cases—being lacking both in volume and promptitude—and he usually attempts to make up for this deficiency by excessive *articulatory* efforts. Any lay pupil can be got to see that just as a violinist would utterly fail to produce any music from his instrument if, neglecting his *bow hand*, he puts his *misdirected* energy into the *string hand*, so the stammerer, forgetting the essential *vocalisation*, must inevitably "stutter" or "stick" in his attempts to produce words by (exaggerated) *articulation* alone. He can thus be got to realise that he stammers because he *bestows too much attention upon articulation and too little upon vocalisation*; the main object, then, in treatment is to emphasise and bring out whenever possible the *vocal* element in words.

The real significance of the fact (previously mentioned) that practically no one ever stammers in *singing* is thus revealed to the pupil, who has usually noted the fact for himself without understanding the reason. It is just because in singing vocalisation *must* be emphasised (and the full vocal value given to words) that articulation presents no difficulty to the stammerer. It is well at the first lesson, then, by insisting on this characteristic feature in stammering, to endeavour to inspire the pupil with confidence in his ability to conquer his difficulty even in ordinary speaking, if he will learn how all-important vocalisation is.

It may be of some interest to mention here another instance of the practical value of emphasising the vocal element in words which was discovered by the writer many years ago. He found that the pronunciation of many well-known stumbling-blocks presented practically no difficulty if the "voice" or "tone" of someone else was made use of during their enunciation; the stammerer's attention was so directed to the production of the correct *sound* that the articulation of the difficult words was as easy as in singing. As pointed out elsewhere,[1] this little "tip" may be useful in many cases (to those stammerers who have the faculty of imitating the voice of others) by enabling them to read aloud (should they be asked to do so) before strangers, who probably will not be aware that the reader is making use of any other than his own natural voice.

The great importance of attention to vocalisation by the stammerer having been sufficiently insisted upon, the pupil should next be taught how to deal with the different letter-sounds which present difficulties to him. For the purpose of tabulating this, Wyllie has drawn up what he terms a Physiological Alphabet, a copy of which, by his kind permission, is here inserted.

[1] *The Practitioner*, January 1903.

A PHYSIOLOGICAL ALPHABET (Wyllie).

I. Vowels.

Y—I E A O U—W

These should be pronounced in the Latin manner as ēē, eh, ah, oh, ōō; y and w are consonants, not vowels, but are placed here for reasons given in the text.

II. Consonants.

	Voiceless Oral	Voiced Oral	Voiced Nasal Resonants
Labials (First Stop-Position)	P (W)	B W	M
Labio-Dentals	F	V	
Linguo-Dentals	Th[1] S	Th[2] Z	
Anterior Linguo-Palatals (Second Stop-Position)	Sh T (L)	Zh D L R	N
Posterior Linguo-Palatals (Third Stop-Position)	K H or Ch	G Y (R)	Ng

The voiceless W and voiceless L have been shown in brackets, the former being now almost confined to Scotland, the latter being peculiar to Wales. The uvular (burring) R is also put in brackets.

It will be well to indicate the main features of his plan in drawing up this alphabet, beginning with the first group of letters.

I. Vowels.

Since all the vowels are essentially produced in the larynx, and are thus full of "voice," they are not grouped in columns as are the consonants. The pupil should be instructed to give the full vocal emphasis to each of them in speaking, but as a general rule it will be found that the vowels are not a source of difficulty to stammerers. Wyllie's reason for placing the two consonants W and Y along with the vowels is interesting, especially to stammerers. It so happens that words commencing with either of these two letters prove very frequently a great source of trouble to pupils who attempt to pronounce them *voicelessly*, the air escaping fruitlessly from their lips. Now, each of them is essentially a *voiced* letter (as shown in the table of consonants), but further, Wyllie points out that an initial W is really (when considered phonetically) equivalent "to a tight ōō with a slight fricative element added," Y being similarly equivalent to ēē with a slightly fricative addition. Thus the words "will you" might be phonetically spelt "ōō-ill ēē-ou," the resemblance to the pure vowel sounds being so great that W and Y may be regarded (as Wyllie suggests) as "stepping-stones" between the vowels and consonants. Now this is not merely a matter of interesting speculation; for the stammerer it gives a lesson of the greatest value. Many very common words have these letters for initials, and the word "yes" alone plays a great part in conversation, and yet it is extremely common to find the stammerer having marked difficulty with it because of his attempts to pronounce the initial letter-sound voicelessly as "yeh." Get the pupil to grasp the fact (and *prove it to himself* by practising) that the word "yes" can be (and *should* be by him) pronounced almost as if in two syllables—"ēē-ess" (with ample vocalisation of the initial ēē), and he will find he has not only got over this oft-recurring stumbling-block, but obtained the key to the enunciation of a whole host of formerly troublesome words, his guiding rule being essentially this, viz.: all words having an initial Y to be pronounced as if they began with ēē; all beginning with W (except those commencing with "wh") to be pronounced as if commencing with ōō.

II. Consonants.

It must be noted at the outset that each of these is considered in the alphabet "as it is pronounced during the enunciation of a syllable containing it"; *e.g.* F is regarded, not as "eff," but the *initial sound* in a word ("fat").

The great importance of recognising the vocal element in the consonants is shown by the manner in which Wyllie separates "voiceless" from the "voiced" letters. This is the essentially important feature in the arrangement of the alphabet so far as concerns the stammerer; the horizontal grouping of the letters by Wyllie does not concern us in this article, though of no little importance in other respects.

It is most important that the pupil should be made to prove to himself (by practising) the fact that, while the larynx takes a very prominent part in the enunciation of each one of the consonants grouped in the second and third columns of the alphabet, those classed as "voiceless oral" have really no laryngeal element of "voice" in them at all, being produced (as the names applied to them indicate) in the oral cavity by the action of the lips, tongue, teeth, palate, etc. upon the air expelled from the lungs. It is this essential distinction which plays such an important part in the training of the stammering pupil, for whom the rule (in dealing with consonants) may be thus summarised: *In speaking or reading, each of the consonants which normally contains "voice" must have its full vocal element given to it; those which are normally voiceless should be touched off as lightly as possible, the voice being brought out with emphasis on the ensuing vowel or voiced consonant.* Perhaps this will be best illustrated by an example, *e.g.* in saying "Tom Bowling" the voiceless "T" is to receive quite a different treatment from the vocalised "B." The initial T is scarcely to be *pronounced* at all; the pupil is merely to apply such compression to the air in his mouth as will communicate an explosive character to the fully voiced residue of the word "-om." The stammerer's natural tendency is to direct his energy to the *initial* letter, but this being a *voiceless* one, it is the ensuing part of the word that he is to concentrate his attention upon—the smallest possible T (as it were) being produced which will suffice to clothe the vocalised fragment and convert it into the word "Tom." On

the other hand, B is a *voiced* consonant: the pupil must therefore throw his voice boldly into its production, emphasising the laryngeal element of it, so that it almost sounds as if it were spelt "ub-owling." The pupil thus must learn to touch off as lightly as possible the voiceless (one might almost call them "articulatory") letters—merely *forming* them without attempting to *sound* them—whereas the voiced initials are to have their vocal element almost exaggerated.

Now, if the teacher will go over *seriatim* the letters in Wyllie's alphabet he will easily manage to demonstrate to the pupils the existence or absence respectively of "voice" in each, and it is most important that the pupil should be made to *prove* to himself the nature of each (by experiment in his teacher's presence), so that he may be made to realise this all-important distinction, and not merely learn off the columns of consonants by rote under their respective headings. It is also easy to show that the intrusion of "voice" into one letter may convert it into another, *e.g.* if "voice" is added in saying the word "pan," it inevitably becomes" "ban," and similarly D, V, Z, and G (hard) are the voiced equivalents of T, F, S, and K respectively. It is well worth spending some little time over this important part of the subject, for it is upon his accurate knowledge of the nature of each consonant (and his compliance with the rule for the treatment of each group) that the success of the pupil's efforts to effect a cure will depend. To test his accuracy, therefore, it is well at each lesson to ask him to point out in any given sentence the first letter in each word on which the voice is to be thrown, *e.g.*, "P*u*t *m*oney *i*n *th*y p*u*rse" (note that "thy" is an instance of the *voiced* "th" as contrasted with the voiceless "th" of such a word as "thin"). Moreover, the teacher should carefully watch during the lessons to see not only that the voiced consonants are properly emphasised, but that the voiceless letters are also reduced (as it were) by the pupil to the smallest possible size, the correct method of dealing with the particular letter being at once pointed out in the event of a difficulty arising.

As I pointed out elsewhere,[1] the teacher ought to be able (by merely *listening*) to tell whether the letter that the stammerer "sticks" at is "voiced" or not; for if the pupil is giving the proper vocal element to each of the voiced consonants, the

[1] *Brit. Med. Journal*, July 20, 1901, p 31.

preliminary laryngeal sound will be audible on their production. Thus, in saying "Pepys' Diary," the formation of the initial letter of the first word is an inaudible act; the explosive character of the succeeding letters is *silently* prepared; but in the enunciation of the next word the pupil should give quite audible evidence that a voiced letter is on its way, the laryngeal element in the "D" being emphasised as though the word were (phonetically) "ud-iary." Teacher and pupil thus are to concentrate their attention on the differentiation of the voiceless and voiced consonants contained in Wyllie's alphabet. A few notes upon some of the letters (with reasons for certain omissions) may be of use in further explanation.

(1) *Voiceless Oral Consonants.*—Each of these (in accordance with the guiding rule given above), when it occurs as an initial, should merely mould the word (give it a shape), so to speak, but in no way be allowed to dominate it, the ensuing "voiced" letter being the part which should receive emphasis. This can be demonstrated to the pupil as follows—the *viva voce* illustration being of course much more efficient than the printed words. Take such a sentence as "Fanny took Kate's top," and teach the pupil at first to practise the emphasising of the *voiced* portion of each word, viz. "-anny -ook -ate's -op." When he has grasped the need of fully sounding these voiced fragments, let him see that the smallest possible enunciation of "F" will convert the vocalised fragment into the complete word "Fanny"; and so on with the others, the voiceless initials are merely to be *enunciated*, the vocalised fragments are to be emphatically *sounded*. In this connection another little "wrinkle" may be of some service. It is a well-recognised fact that it is especially when occurring as *initials* that the voiceless consonants prove troublesome to stammerers. Now, in the sample sentence given above it may be seen that the third word ("Kate") commences with the terminal letter of the preceding word. Since the voiceless K has just issued from the pupil's mouth, there is no real need for him to reproduce it to "clothe" the fragment "-ate's"; he merely requires to tack on the terminal "k" to the succeeding vocalised syllable, as if the sentence ran thus, "Fanny took-ate's top"—no pause, of course, to be allowed at the end of the word "took." This simple procedure, then, may be put thus as a working rule: "*When the voiceless initial of any word in a*

sentence is the terminal letter of the immediately preceding word, no attempt need be made to reproduce it as an initial." The practice enjoined of "touching off lightly" the voiceless initial is thus carried to its fullest possible extent, the voiceless initial being practically omitted.

It will be noticed that the letter C does not appear in Wyllie's alphabet—where equivalent to "soft" C (as in "city") it is represented by S; where "hard," it really represents K (as in "cat"). Wyllie regards H as a "voiceless fricative of the posterior stop-position," and certainly on analysing the phenomenon of its production it is closely analogous to the terminal soft "ch" in the word "loch" (as a Scot would pronounce it). This is fairly well shown by comparing the *sound* produced by the pronunciation of the words "Law he" with the correct spelling of the actual place, viz. "Lochee."

(2) *Voiced Oral Consonants.*—Sufficient stress has already been laid on the need for full vocalisation of each of these, but certain letters call for notice. Thus the compound letter-sound "zh" really represents the *sound* given to the letter "s" in such words as "pleasure"; it never occurs as an initial.

L and R very frequently prove troublesome to a stammerer, probably because of the fact that each has a peculiar trilling action as part of its *articulatory* mechanism—in the first case the sides of the tongue being brought into play; in the second its tip. Here again the pupil is so absorbed with this *articulatory* feature that he usually entirely neglects the more essential and primary *vocal* element present in each; they are both *voiced* fricatives, and must therefore both be thoroughly vocalised.

The manner in which W should be dealt with (*i.e.* as "a tight ōō") has been laid down when speaking of the vowels, but another important point can be best noticed here. It will be found that usually words which commence with *two or more* consonants are particularly difficult of pronunciation by stammerers, as if the combination of the two consonantal sounds (perhaps one voiceless and the other voiced) overtaxed the already defective "gearing" of the speech mechanism. Now, there are several very common words commencing with "wh," and it may be well to indicate how these stumbling-blocks can be surmounted. The "w," of course, represents the vowel "ōō," and is to be sounded as such; but the pupil must further note

that in pronouncing such words the "h" really phonetically *precedes* the "w," so that the word "when" is to be pronounced as if it were spelt (phonetically) "hōō-en," and so with all similar words. The stammerer almost always tries to produce the initial "wh" of such words *voicelessly* and fails; let him learn to vocalise the "ōō" sound of the "w" thoroughly (making almost two syllables of such words as "hoo-ich," "hoo-ile"), and he will find he can enunciate his former stumbling-blocks with ease. In like fashion words commencing with "tw" (*e.g.* twenty) and "sw" will give no trouble if the "w" is fully sounded as "ōō," *e.g.* "sōō-eep" (sweep). The importance of fully vocalising an initial Y has already been insisted upon.

(3) *Voiced Nasal Resonants.*—The letter sound "ng" never occurs as an initial so no further reference to it is required here, but the pupil must have firmly impressed upon him the importance of recognising that M and N are each "voiced" letters, for it is extremely common to find stammerers trying to produce these initial letters voicelessly. Each should be produced with a loud emphatic hum (which, as it were, heralds the production of the word), so that *e.g.* the words "North Moor" should be pronounced as if they were spelt (phonetically) "(un)north" "(um)moor."

A short reference is necessary, before passing from the subject of Wyllie's alphabet, to the omission from it of certain letters. The absence of C has previously been alluded to; Q is unnecessary since it is equivalent to "kw," just as X is in like fashion represented by "ks" (compare the sound of "Styx" and "sticks"). The letter J calls for a fuller notice. As is pointed out by Wyllie, this is really a compound letter-sound equivalent to "dzh," which is also the phonetic spelling of G "soft," as is shown in such a word as "Jill," which has the exact pronunciation of the Scots measure a "gill." It is very important, therefore, that the pupil thoroughly understand that any word commencing with "soft" G or J is to be considered as commencing really with the letter D, and so must receive the ample vocalisation due to that letter.

In the course of the notes upon the Physiological Alphabet, most of the chief difficulties of stammerers have been dealt with, but a few more points may be usefully alluded to here before summarising the treatment as a whole. The difficulty

of dealing with *two* (or *more*) initial consonants has been mentioned already, but a fuller reference to it here may be helpful. When the initial letter is *voiceless*, and the succeeding consonant *voiced*—as in words like "train," "flow," etc.—the pupil must be taught first to emphasise the voiced portion only, viz. "-rain," "-low," and then practise inserting before these the real initial letter of the word, almost inaudibly. For instance, in saying such a word as "proud," once the pupil has learned the need of emphasising the *vocal* portion ("-roud") he must be taught that the very slightest apposition of the lips is all that is required to formulate the initial "p"—*not* a tight pursing of the lips, as is so apt to be the case with stammerers (with a consequent "bottling up" of the word in the oral cavity), but merely (if the phrase be permitted) the thinnest possible (almost diaphanous) garment to clothe the naked form behind it. Still more is this minimising of the voiceless initials required when a word has occasionally *two* such letters preceding the voiced portion, *e.g.* "strike."

On the other hand, trouble is sometimes apt to arise in the case of words having *two voiced* initial consonants (as in words commencing with "gl," "gr," "dr," etc.), either owing to the fact of the stammerer omitting to vocalise the primary initial consonant, or perhaps oftener his attempt to produce the two consonants almost simultaneously, with consequent articulatory difficulty. He must be taught to throw his voice boldly into the *primary* initial in such words, and usually the remainder of the word will then follow easily, containing, as it often does, nothing but voiced letters, *e.g.* "drown," "gleam." If such words, however, prove very troublesome (as is often the case), then it is well (at least in the early stages) to get the pupil to practise emphasising the initial letter to such an extent as almost to make it a syllable by itself, as if the word were spelt (phonetically) "(u)d-rown," "(u)g-leam," etc.

Enough has been said, I think, to emphasise and expiscate the points in the training of the pupil in the use of the Physiological Alphabet. It may be helpful now to summarise the course to be adopted in undertaking the treatment of a stammering pupil before alluding to one or two final points.

It is essential at the outset (in his first conversation with the pupil) for the physician to notice what is the form of

stammer manifested by his patient (who will probably, of course, be at his worst under such a nervous ordeal), and in the great majority of cases it will be found to present the type described above, viz. a lack of amount and promptitude in the vocalisation of consonants, with a tendency to exaggerated articulatory efforts, especially in producing the voiceless explosives.

The first lesson to be given, then, to the pupil is an explanation of the *twofold* nature of the process in speech-production (vocalisation and articulation), and with the help of Wyllie's simile of the violin-player even young pupils can easily be got to realise the part played by each of these two functions in speaking. Nor is it difficult to get them to realise how greatly adequate attention to vocalisation will help them, for nearly always these pupils have noticed that they are free from their difficulty when *singing*, so that it is only necessary to explain to them the *reason* of this facility in singing of those words which prove so troublesome in talking. Should he be so devoid of a "musical ear" as to be unable to sing, then occasionally the writer's suggestion as to the adoption of the "tone of voice" of another may be feasible, in so far at least as to convince him of the fact that greater attention to vocalisation (with less attention to articulation) is to be his means of conquering his difficulty. Another method of demonstrating to the pupil the important part played by vocalisation is to get him to read some of the sentences of the Prayer-Book in the style and "voice" used by the Anglican clergy in reading the service. He will nearly always be able to enunciate easily in this fashion words having initials which cause him difficulty at other times, provided he pays sufficient attention to his intoning. Thus, it is well known that Charles Kingsley stammered badly in ordinary conversation, though able to conduct the service and even preach with little hesitancy of speech. Moreover, it must be especially demonstrated to him that no exaggeration of articulation can ever make up for defective vocalisation. Next Wyllie's alphabet should be given to him and explained. The writer is in the habit of devoting at least three or four lessons (of about an hour each) to this all-important part of the subject. As mentioned above, it is of the utmost importance that the pupil should not only understand but *prove* to himself (by repeating before his teacher) the existence or absence of "voice" in each letter therein

contained, for this is the key to his difficulty, and he must be able to point out at once the first "voiced" letter in any word from him. With a view to fixing the recollection of the voiced and voiceless letters respectively in the pupil's memory, Prof. Wyllie has drawn up a set of sentences for the use of stammerers, reproducing the consonants in groups according as they are voiced or not. These sentences by his kind permission are here inserted.

A. *Initials that contain voice.* (In the pronunciation of these the voice is to be thrown boldly into the initial.)

1. "Even ancient elves are awed over oozing."
2. "We visit the Zulus like ramblers yearly."
3. "Best gold dust."
4. "My nephew."

B. *Initials that do not contain voice.* (These are to be touched off lightly, the voice being brought out promptly in the ensuing vowel or voiced consonant.)

1. "Far shores seem thinly hazy."
2. "Two poor comrades."

It is often advisable to give additional sentences for practice upon the specially difficult letters in each pupil's case, and until the patient has got quite familiar with the distinctive method of dealing with voiceless and voiced consonants respectively, it is well to restrict the sentences to the one class of consonants, so that he may not be confused by the change required suddenly to produce the others correctly. Thus to give a few illustrations :—

A. *Voiced*—

"Davy Doldrum dreamt he drove a dragon."
"Gaffer Gilpin got a goose and gander."
"Mother make more mustard ; no, no, not now."

B. *Voiceless*—

"Five fat friars fanning fainting flies."
"Two teetotums totally tired trying to trot to Tadbury."
"Peter Piper picked a peck of pepper."

The pupil can, of course, easily draw up sentences containing the specially difficult initials for his own practice daily, and when he is familiar with the proper treatment of each class of consonants, each sentence should contain voiceless and voiced initial consonants, so that he may acquire the habit of promptly touching off the former and emphasising the latter. I have

found it often very helpful in gaining the patient's confidence in my ability to help him—a matter of no little moment in the treatment of a neurosis—to show to him even at the first lesson how such a common word, *e.g.* as "Yes" (which very often causes difficulty) can be readily enunciated by him by the emphasising of the vocal element in the initial. The pupil thus gets an insight into the *practical* side of the treatment, which will encourage him in the process—to him at first sight rather elementary—of learning anew the alphabet again.

Special attention should be paid, of course (as given above with full details), to the method of dealing with the more troublesome letters L, R, J, etc., the management of W and Y, and particularly the proper enunciation of words commencing with "wh," as illustrated in such a sentence as—"When will you walk yonder?" which he is to pronounce thus—"hōō-en ōō-ill ēē-ou ōō-alk ēē-onder?" It is often useful to note the words which cause the patient most difficulty in reading or talking, and get him to practise these by forming them into sentences to be repeated at his subsequent lessons.

In all his lessons—his practising at home as well as those in presence of his teacher—the patient should *stand up* and speak out in a full (not necessarily loud) voice, and he should also follow Wyllie's suggestion of "making music with the voice, and *listening* to it as he speaks," *i.e.* cultivate the habit of attending specially to his *vocalisation*.

Undoubtedly another extremely important point to attend to is the cultivation of the habit of keeping always an ample supply of air in the chest to enable the patient to fully vocalise each appropriate letter. The tendency to speak—or attempt to speak—from a relatively "empty chest" is very common amongst stammerers, largely, no doubt, because so much of their breath is fruitlessly wasted in trying to produce orally letters which should originate in the larynx. In some cases, however, there is also deficiency in the expansion of the lungs from faulty methods of respiration, so that in such instances there is not merely a waste of air but a deficient store of it to begin with. Exercises, therefore, to improve the capacity of the chest as well as the control of the exit of air from the lungs will be found advantageous in most cases. It is scarcely necessary to mention that the pupil should constantly endeavour to avoid speaking abruptly

or hurriedly, cultivating instead a deliberate (but not monotonous) utterance.

Each of the pupil's daily lessons should commence with the reading of Wyllie's stammerer's sentences given above, followed by careful practising of additional sentences (drawn up at first by his teacher, later by himself) containing exercises upon the consonants which especially cause him difficulty, and particularly any words which prove stumbling-blocks to him in his daily conversation (as noted by his teacher or himself). The next stage is to advance him to the reading aloud—perhaps *enunciating* is the better descriptive term—of poetry; the simpler forms, such as Scott's, do admirably, especially some of the songs, *e.g.* in "Lady of the Lake," to take just a couplet in illustration of the way in which the writer tries to emphasise the vocal element in the voiced words, while the voiceless initials are "filed down" (as it were) to the smallest possible size. Thus—

"He is gone on the mountain,
He is lost to the forest,"

is to be enunciated as if spelt phonetically—

"He is (u)gone on the (um)mountain,
He is (ul)lost‿oo the forest."

Note how the *terminal* "t" of "lost" is made to act as *initial* for the vocalised fragment of the succeeding word as explained previously. It is not long before the pupil learns the art of looking ahead, as it were, for the voiced letters upon which his energies are to be chiefly expended, and if the lines are spoken *slowly* and *impressively* it is quite possible to *emphasise* the vocal elements without *exaggerating* them. Blank verse may later be tried, and last of all prose as reading exercises.

When the pupil is going over his lessons with his teacher, any stumbling or error of enunciation should, of course, be noted by the latter and the proper method of producing the difficult consonant explained. In this connection it may be mentioned that in nearly every case it will be found that the pupil is much worse at certain times than at others; if he shows signs of being worse than usual at his lesson—stumbling at words which formerly caused him little trouble—it is usually advisable to discontinue the lesson promptly, for his unsatisfactory efforts will only make matters worse owing to nervousness. It may perhaps

be not amiss to indicate the time ordinarily devoted to the pupil by the writer in treating cases. Usually I get him to come on successive days (for about an hour) till the whole of Wyllie's alphabet, etc., has been explained—say five or six lessons in all. Then he is asked to return twice weekly for a fortnight to repeat his lessons and allow of observation on his carrying out the instructions given. Thereafter a monthly practice for about half an hour before his teacher for six months or so is usually sufficient to keep him on the right lines towards recovery, provided the pupil is earnest in his desire to be cured, and persistent in his home practisings of about half an hour daily.

This last point is the real crux of the whole matter. Some stammerers are indifferent to their lot; often, no doubt, as the result of fruitless attempts at so-called "cures"; these pupils it will be very difficult to cure, for patient persistence in practising is the essential above all others in overcoming their difficulty. It should be remembered that it is not only the learning of the *correct* method of enunciation which is required, but in many cases there is also the unlearning of the *faulty* method (which comes so naturally from long usage), and this is even more difficult, or at least calls for more watchfulness on the pupil's part. On the other hand, a pupil who is eager to master his difficulty, and willing, therefore, to *think* before he enunciates (and not let the words come out anyhow), and practise *with persistence* on the lines laid down for him, will find not merely marked improvement result therefrom as the weeks pass into months, but at an increasing rate as his sense of being able to conquer his former difficulties thereby lessens his previous nervousness. The cure of such a deep-rooted (though functional) affection is by no means easy, but the chief difficulty is not in getting the pupil to *understand* the treatment, but in getting him to carry this out perseveringly.

No definite rule can be laid down with regard to the *age* at which treatment should be begun. While it may not be easy to get young pupils to *understand* exactly the physiological principle upon which Wyllie's treatment is based, his simile of the violin-player to illustrate the mechanism of speaking can be understood by children; the really important question is the pupil's earnestness in desiring to master his difficulty, and his willingness to persist in his lessons—a matter more of personal characteristics

than of age in most patients. While it will be gathered from the above remarks that the prognosis in each case depends mainly on the will-power and determination to practise of the particular pupil, the writer does not by any means share in the serious views of some who would almost look upon stammering as an affection tending to shorten life from the strain upon the nervous system. Like many other functional conditions it may prove somewhat intractable, but surely it is very rarely fatal.

At the outset I stated that Wyllie's method of treatment of stammering was of such a nature as rendered it equally suitable for the general practitioner and the specialist; in the somewhat lengthy account given of it above I trust that description has not been belied. What is called for, however, I venture to think, is a wider recognition by teachers of medicine of the fact that stammering is not merely a condition within the purview of our profession, but a neurosis which any practitioner ought to be competent to treat upon scientific lines, instruction in which should form a part of his undergraduate clinical and systematic training.

In conclusion, the subject of *prophylaxis* calls for a brief notice, viz. the question whether a pupil who stammers badly should be educated along with other boys, as in a public school. There is no doubt that in several instances stammering is largely acquired by a process of imitation, so that in this view the stammerer is a source of danger to others; while, as regards his own comfort and happiness, the presence of a crowd of school-mates—not by any means always sympathetic—is too often a source of aggravation of his neurosis. There is, moreover, the further practical point, that in much of the class teaching prompt and *hurried* answering by the pupils is necessarily called for, the very conditions which will increase the stammerer's nervousness and impediment. If the pupil is trying to carry out at home (under the supervision of his medical attendant) the physiological methods described above for overcoming his neurosis, the good effects of this will be largely neutralised by the hurried (often competitive) *viva voce* work at school under conditions which make it well-nigh impossible for him to follow out properly the correct methods of utterance. In some cases, therefore, especially where the pupil is unusually sensitive and shy, or the impediment is exceptionally severe, it will probably be better for the

pupil (and his class-mates) that he should be taken from school and placed under the care of a tutor, who, in addition to carrying on his general education, can supervise the physiological treatment of his stammering under the instructions of the medical adviser upon the above lines.

RETURN OF PARAPLEGIA IN A CASE OF OLD INFANTILE PARALYSIS.

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On the 21st of January 1907, there was brought to the Hôtel-Dieu, bed No. 20, in the salle Saint-Christophe, a man of forty years of age, an accountant by profession, who had lost all power of walking. Some minutes before he had been walking on the Boulevard St Michel, when he was suddenly seized with weakness in the legs, which caused him to fall in a heap. He was brought to hospital, and at the present time it is still impossible for him to move the lower limbs.

On uncovering the patient and examining his legs, we are immediately struck with the diminution in their size, with the muscular atrophy of the limbs from the knee; this is a kind of *atrophie en jarretière* which affects specially the gemellus, peroneal, and antero-external muscles of the leg, and the tibialis anticus muscle in particular. This atrophy is not limited to the muscles; it affects the bones also, and palpation of the tibiæ shows that their dimensions are smaller than normal. Moreover, the tibia of the right side presents a callus, which corresponds to a cutaneous cicatrix. The callus and the cicatrix are both the results of a compound fracture which he had sustained thirteen years before from the fall of a heavy weight on his leg. The feet are deviated and deformed; they turn inwards; the great toe bends inside the middle line and tends to approach the posterior part of the foot. The anterior portion of the foot droops, and in walking the patient leans upon this anterior part, and upon the outside edge of his foot. The sole is hollowed out into a well-curved arch, and this plantar arch has to be sustained

in walking by orthopedic shoes. The deformity is what is known as equino-varus club-foot. Attempts have already been made to straighten the foot, for when patient was ten years old, section of both Achilles tendons was performed. The scars of this operation are still visible.

Examination of the tendon reflexes does not reveal any disturbance of the patellar reflexes, which are almost as marked as normally; but the Achilles reflexes are diminished. Diminution of the reflexes would thus appear to be limited to the areas of muscular atrophy. There is no foot-clonus. Examination of the plantar reflex causes no movement of the toes; there is no Babinski sign.

Sensibility is absolutely intact. There are no sphincter troubles. The trophic or vaso-motor troubles are very slight; they consist merely in transitory perspirations, or in sensations of cold in the feet.

The patient has always been aware of these deformities of his feet; he says that they date from his infancy, that in the first months of his life he suffered from convulsions, then from paralysis. His convalescence was slow, and for a long time he had to walk with some orthopedic apparatus, which was at first applied to the whole of his lower limbs and the waist, then to the legs and thighs, then to the legs alone, and finally, when he was about fourteen, he could come and go without the aid of any apparatus.

The case was evidently one of an infantile atrophic paralysis. The site of the paralysis, the integrity of the reflexes, and the absence of any spasmodic phenomena lead us to the diagnosis of *infantile spinal paralysis.*

The objective signs of this paralysis, and the evolution which we have just recorded, do not in themselves present any interesting peculiarity.

On the other hand, the history of the patient from the age of fourteen seems worthy of note. Contrary to what is observed in most cases of infantile paralysis, where the disease once terminated leaves only some infirmity without any morbid return, we have here a series of paralytic accidents in the lower limbs which have re-appeared during the last twenty-two years.

At the age of 18 (the patient could walk without any

artificial support since the age of 4), he found one day, on trying to rise from the table, that his legs would not carry him; he was put to bed and for four months his lower limbs were paralysed. He does not seem to have had any fever. The paralysis did not extend to other muscular territories, and the atrophy of the muscles affected in childhood was not aggravated. After this period of four months, patient felt some tingling in his legs, and gradually began to walk again.

At the age of 22 (four years later) one evening on returning from the theatre, he fell on the Boulevard Sébastopol; he was put into a conveyance and taken home: there he was put to bed, where he remained for three months in the same condition as on the former occasion. Again he commenced to walk. At the age of 25, on coming from his work, he fell in the Avenue de l'Opéra; he was taken home, and lay paralysed in bed for two or three months.

At the age of 29, whilst walking one day on the Boulevard St Michel, his legs gave way under him: he was taken to the Hôtel Dieu, salle St Denis, and there he remained for six weeks, paralysed.

At the age of 35 he was in Rouen; he felt unwell one evening, went to bed, and had fever. Next morning, on trying to rise, he fell on the floor. He was put back to bed, and the fever continued and lasted for three weeks: he had, he says, mucous fever.

During the convalescence from this fever, he attempted to rise; but all movement of the lower limbs was impossible. He remained paraplegic for eight months, and then gradually regained power of walking.

At the age of 39, in October 1906—that is, three months ago—he fell on the Place Maubert and was taken to the Hôpital de la Pitié, into M. Rénon's ward, and there he remained three weeks.

In November 1906, some weeks later, he again fell down in the Boulevard St Germain; again he was taken to La Pitié, where he remained fifteen days.

Finally, quite recently, at the commencement of the month of January, he fell down in the Rue de Rennes; he was taken to La Charité, into M. Toupet's ward, where he remained until some days ago. When he left hospital, he was walking almost

quite well ; but again his legs gave way on the 21st of January, which necessitated his being brought to the Hôtel Dieu.

From the age of 18 to that of 40, that is to say, in the space of twenty-two years, this patient who had been the subject of an old spinal paralysis, has thus been affected by nine returns of attacks of paraplegia, which have been transient, and not accompanied by fever, and which have been neither accompanied nor followed by any progress of the paralysis.

The case is, without doubt, one of spinal accidents resulting from the infantile paralysis.

These tardy spinal accidents of acute anterior poliomyelitis are now well known. There is no longer any question of regarding this affection as a simple infirmity without any ulterior effect, related to a definite cicatrix of an inflammatory focus in the cord. We must refrain from concluding with Ross, "that subjects who have been affected by infantile paralysis do not really seem more exposed during the course of their existence to other affections of the cord, than those till then free from any internal lesion."

Indeed, this writer's views have been contradicted by the observations and works of numerous authors. Raymond published in 1875 the first observation of progressive muscular atrophy following infantile paralysis. Then came the works of Hayem, Quinquaud, Coudoin, Seeligmüller, Oulmont and Neumann, Sauze, Landouzy, and Déjerine.

The paper by Ballet and Dutil, and the thesis of Sattler, have brought the question to a point, and have established a definite classification of these spinal accidents.

We might mention also the later publications of Grandou and of Bernheim, the clinique which Brissaud has devoted to late muscular atrophies following infantile paralysis, and finally the work of Pierre Marie on late scoliosis in infantile paralysis.

Taking into consideration all the facts to which we have alluded, we may, with Ballet and Dutil, classify the late spinal accidents after infantile paralysis in the following way :—

(*a*) Congestive attacks, with paresis or transient paralysis.

(*b*) Acute myelitis in the form of acute adult spinal paralysis.

(*c*) Sub-acute myelitis in the form of sub-acute anterior spinal general paralysis.

(*d*) Chronic myelitis in the form of progressive muscular atrophy. The statement of this classification at once makes it evident that our observation belongs to the type of "congestive attacks with paresis or transient paralysis."

Leaving aside, then, those spinal accidents which assume the type of acute spinal paralysis of the adult, of sub-acute generalised anterior spinal paralysis, and of progressive muscular atrophy, we shall confine ourselves to some considerations with regard to the first type of paresis or transient paralysis superposed upon an old infantile paralysis.

This type is described by Ballet and Dutil in the following terms: "An individual bearing the indelible stigmata of an old infantile paralysis, having, for example, one slender and atrophied lower limb, but experiencing on account of this infirmity merely a more or less marked awkwardness in walking, which renders it difficult and less secure, but does not completely impede it, is suddenly affected, without apparent cause, or under the influence of cold, by complete or incomplete paralysis of the lower limbs. This paralysis is transient, and disappears after some hours or some days, but may not impossibly return later."

These writers quote in support of their description a personal observation, certain details of which are characteristic: it is the history of a man of 34 years of age, who was affected in the early years of his infancy by a paralysis of the lower limbs. His limbs were atrophied and showed vaso-motor troubles. In spite of this paralysis he could take somewhat long walks, and during the siege of Paris and the Commune he figured amongst the combatants. But in 1871 (patient was then 22 years of age, and was a prisoner at Versailles on account of the affairs of the Commune), he found one morning that he could not raise himself out of bed. He remained in this condition for a whole day. Already, in the preceding winter, on coming out of his office, he had suddenly experienced great difficulty in standing. From that time until the age of 25, this weakness of the legs became so marked every winter as to render walking exceedingly difficult. In order to get along, patient was then obliged to lean upon a chair, which he pushed in front of him. When spring-time came, the strength partly returned to his lower limbs.

In this case we have evidently a syndrome analogous to that presented by our patient. But our case appears to be still more

characteristic, as the patient has had nine attacks of paraplegia. Moreover, in his case the attacks of paraplegia lasted several weeks or several months, whilst Ballet and Dutil allude merely to paraplegia of some hours' or of some days' duration. The anatomical basis of these attacks of paraplegia has never been ascertained, and it is only by analogy with certain medullary congestive phenomena, such as the œdema and congestion of transient Pott's paraplegia, that we can relate those transient paralytic troubles to a congestive process taking place in the circumference, or in the neighbourhood of the old cicatricial focus.

But whatever may be the value of this interpretation, the clinical fact none the less remains, and it will be of service to recognise it, in order that we may be able to look forward to the satisfactory evolution of a paraplegia occurring suddenly in an infantile paraplegic.

Still, though the prognosis is not grave, and though the paralytic troubles may easily and rapidly disappear, it is none the less true that after a certain number of returns of paraplegia the weakness of the lower limbs may become persistent. In our case it became gradually established, and in the intervals between the attacks the patient no longer recovered unimpaired his power of walking. The patient of Ballet and Dutil, three years after his attack of paraplegia, was seized one morning on rising by a complete paralysis of the lower limbs, which still persisted eight years later, with the addition of atrophy and progressive enfeeblement of the muscles of the lower limbs.

So, then, we may agree with Sattler that successive inflammations, by diminishing the vital resistance of a cord already not uninjured, will by so much increase its morbid receptivity. It prepares the way for other more serious local affections, which will find, in the enfeeblement of the patient, a condition exceedingly favourable for their evolution.

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Abstracts

ANATOMY.

CYTOLOGY AND DEVELOPMENT OF THE NERVE CELLS.

(134) **(Recherches cytologiques sur le développement de la cellule nerveuse.)** REMY COLLIN, *Le Nevraxe*, Vol. viii., Jan. 1907, p. 185.

THIS elaborate memoir (extending to 123 pages) proves of particular interest to the writer, as it has appeared almost contemporaneously with a paper by him, dealing extensively with the same theme. It is remarkable to find how closely the main results of these two papers correspond. Collin's observations are founded almost entirely on an examination of chick-embryos.

One of the most important observations is the high degree of metabolic activity which he finds manifested by the nuclei of the neuroblasts. This activity is shown by a pronounced disturbance of the nuclear chromatin, a fact which the writer has also recorded in the chick. Collin describes two kinds of nucleoli in the neuroblast-nucleus—chromatic and plasmatic. The chromatic nucleolus tends to divide into two smaller bodies, this phenomenon being frequently accompanied by a corresponding invagination of the nuclear membrane, which reminds one of an attempt at amitosis. The plasmatic nucleolus is not present in the neuroblast-nucleus from the first. Its date of appearance is variable, and this begins in the form of a small acidophile particle which gradually enlarges. The author has been unable to decide definitely upon the real significance of this body.

Collin also describes the presence of a large number of fine granules in the nucleus during the early stages. He calls these *chromatic microcaryosomes*. The chromatic nucleoli are more or

less spherical during the early stages, but tend later to break up into *spherules* which scatter throughout the nucleus. These spherules group themselves into grotesque patterns, some of which remind one of the phenomena of karyokinesis. The various kinds of particles which are met with in the neuroblast-nucleus may be divided into those which are basophile and those which are acidophile. Collin finds that the basophile material is discharged from the nucleus to form the Nissl bodies, while that which is acidophile in nature, always retains its position within the nucleus. This observation is confirmatory of Scott's recent work on this subject.

A large amount of evidence has recently been brought forward by Held, Rohde, Holmgren, Scott and the writer to show that the material constituting the Nissl bodies is, in part at least, a derivative from the cell-nucleus. This may be termed the nuclear theory of the genesis of these structures, in contradistinction to the usually accepted cytoplasmic theory. The results of Collin's researches go to support the nuclear theory, for he describes the shedding of the basophile nuclear material into the cytoplasm in the form of the above granules to form the Nissl bodies. This is somewhat similar to the results of Rohde and Holmgren, but rather different from those of Scott and the writer, who hold that the nuclear derivative is not discharged as solid particles, but in the form of a fluid substance. The writer finds that the latter is achromatic in nature, afterwards becoming rechromatised outside the nucleus to form the Nissl bodies.

Collin studied the neurofibrillary network, chiefly by means of the Cajal silver method, which in his hands appears to have yielded very satisfactory results. In this way he has been in a position to controvert Bethe's statement that the long peripheral fibrils of the dendrons are distinct from the general network in the body of the cell. Collin describes the two systems of fibrils as blending freely with one another. The endocellular neurofibrillary network is developed before the Nissl bodies, according to Collin. He suggests a possible nuclear origin for this network, but as yet is not definitely decided on this point.

Fragnito, Capobianco, Pighini, and other Italian neurologists have described in the chick, and also in other vertebrate types, the occurrence of a fusion of the nuclei of what they term the *secondary neuroblasts* to form the cytoplasm of the future nerve cell. Collin, like the writer, has been unable to satisfy himself of the existence of this phenomenon. If one makes a series of transverse sections of the spinal cord of the chick at any stage (say the tenth day), and stains these with aniline dyes, such as toluidine blue, methylene blue, or thionine, then the nuclei appear to have an indistinct contour, which suggests a process of fusing

of these elements. If, however, another series of sections from the same specimen be stained by iron-alum-hæmatoxylin (as employed by both Collin and the writer), then the various nuclei will be found to possess a sharp outline, thus preserving their individuality. The latter stain is undoubtedly the best to employ for studying the nuclei of the neuroblasts.

Collin dismisses as untenable the theory which Kronthal has lately brought forward, regarding the development of nerve cells from the fusion of leucocytes. Very few embryologists will feel inclined to tender their support to Kronthal's idea of nerve cell genesis.

The numerous figures illustrating Collin's paper have been very well executed. An extensive bibliography is appended. On the whole, the contents of this memoir will be found to constitute a distinct advance in our knowledge of the ontogeny of the nerve cell. JOHN CAMERON.

THE CENTRAL TERMINATION OF THE COCHLEAR NERVE.

(135) **(La Terminaison centrale des Nerfs sensibles périphériques. Le Nerf Cochléaire.)** VAN GEHUCHTEN, *Le Nevraxe*, Jan. 1907, p. 127.

VERY little work has been devoted to determining accurately the central termination of the cochlear nerve. Only Thomas, Weigner, Wyrubow and Tricomi-Allegra have used Marchi's method for this purpose, and they all found degeneration in tracts which have been generally regarded as belonging to the secondary acoustic system, *i.e.* in the corpus trapezoides, striæ medullares, the superior olives and the paraolivary nuclei. Van Gehuchten, who will not accept their conclusions, argues that degeneration is found here only because on intracranial section of the eighth nerve the brain stem is also invariably damaged, and partly owing to the fact that when the facial nerve is injured so intensely that acute retrograde degeneration occurs in its central end evidence of degeneration is always found in the corpus trapezoides and the other structures through which the nerve passes on its intra-medullary course.

His own investigations consisted of the isolated destruction of the cochlear ganglion in the internal ear in guinea-pigs and the examination of the brain-stem after a suitable interval of time by Marchi's method. In this way he was able to obtain degeneration of all the fibres of the cochlear root without injury to any other structure. He found that all these root fibres terminate in the tuberculum acusticum and the ventral nucleus, and that none of the root fibres extend into the corpus trapezoides, the striæ medullares, or reach the superior olives. GORDON HOLMES.

THE INFERIOR LONGITUDINAL BUNDLE AND THE CENTRAL OPTIC BUNDLE. (Le faisceau longitudinal inférieur et le faisceau optique central.) LASALLE ARCHAMBAULT, *Nouv. Icon. de la Salpêtrière*, Nov.-Dec. 1906, p. 561.

(136)

IN a previous paper (reviewed in this Journal, Vol. iv. p. 31) the author recorded the results of his investigations, according to which the anterior end of the so-called inferior longitudinal bundle, or better the external sagittal layer of the temporo-occipital lobe, consists of a certain number of association fibres, plus the central optic tract or the geniculo-calcarine fibres, which represent the cerebral path of visual impulses; while its posterior portion in the occipital lobe contains practically only geniculo-calcarine fibres.

These conclusions were attacked by the Dejerines, and the present paper is a reply to their criticisms. He first points out that his conclusions have been misinterpreted by his critics, and secondly, he argues that cases which have been published by the Déjerines are confirmatory of his views. He insists that in every case in which a lesion destroys the primary optic centres or the retrolenticular segment of the internal capsule, secondary degeneration is found in the occipital lobe only in the external sagittal layer, in which consequently the corticopetal fibres must be contained; they terminate along the lips of the calcarine fissure. Another point of interest is that, according to the author, the connection between the occipital cortex and the external geniculate body is chiefly by corticopetal fibres (geniculo-calcarine, *i.e.* the true optic radations), while that of the occipital cortex with the pulvinar is almost entirely by corticofugal fibres (thalamic radations). GORDON HOLMES.

THE NEURONE THEORY AND PRIORITY. (Zur Wahrung meiner Priorität in Sachen der Kontinuitätslehre des Centralnervensystems.) B. HALLER, *Neurolog. Centralbl.*, 1907, p. 118.

(137)

UNTIL the introduction of Golgi's impregnation method, the doctrine of continuity of the nerve elements had been accepted by most investigators. Golgi, on the strength of his silver preparations, proclaimed the central nerve-network to be a myth, though later, in 1894, he was less dogmatic on the subject. "The misleading results of the Golgi method," says Haller, "were largely due to the ignoring of other staining methods." As a consequence, the Neurone theory sprang into existence, and, for a time, dominated the field of neurology. Kölliker, the chief supporter of the Neurone theory, maintained the doctrine of neurones anatomically inde-

pendent, in contact with each other but not in continuity. Now the swing of the pendulum is reversed, and many of the former most vociferous supporters of the Neurone theory have become its destructive critics, notably Nissl. Haller complains that the credit of the theory of continuity of nerve elements belongs to himself, and not, as Pflüger has stated, to Apáthy. In conclusion, Haller gives a list of his own writings to prove his claim to priority. PURVES STEWART.

PHYSIOLOGY.

(138) **ON THE FIRST MANIFESTATION OF NERVOUS FUNCTION IN THE LIFE OF THE EMBRYO VERTEBRATE. (Sur les premières Manifestations de la Fonction nerveuse dans la Vie embryonnaire des Vertébrés.)** GIACOMO PIGHINI, *Le Nevraxe*, Vol. viii., Jan. 1907, p. 177.

THE results which are embodied in this brief note are founded on a study of the movements executed by developing embryos of *Pristiurus*, *Scyllium*, *Mustelus* and *Torpedo*, which were kept under observation by the author at the Zoological Station, Naples.

Pighini finds that the first spontaneous movements are observable when the embryo is about 25 mm. long. These become very free when the embryo has attained a length of 30 mm. At the stage when these movements are first exhibited Pighini finds that the central nervous system of these animals consists of a complicated network formed by chains of neuroblasts, while at the same period the multicellular character of the peripheral nerves is also very evident. The embryonic nervous system of selachians at these stages of development thus presents the type of nerve network met with in the coelenterata and echinodermata. The author points out that the nervous functions exhibited at these early stages may be considered as the expression of the activity of a nervous system, which has not yet attained to the type of structure met with in adult vertebrates. The contents of this all too brief paper afford strong support to the multicellular theory of development of the neuron. JOHN CAMERON.

(139) **THE BEHAVIOUR OF SOME REFLEX PHENOMENA AFTER SECTION OF THE POSTERIOR NERVE ROOTS. (Comportamento di alcuni fenomeni riflessi dopo la sezione delle radici posteriori.)** OTTORINO ROSSI, *Rivist. di Pat. nerv. e ment.*, Jan. 1907, Vol. xii., p. 1.

AFTER exposure of the cord in the dorsal region, stimulation of a posterior root excites generalised reflex movements with groaning of the animal. If now one root be cut—in an animal that

is not anæsthetised—and the peripheral nerve be stimulated, similar movements occur not only in the muscles corresponding to the nerve but over the whole body and the animal groans just as before.

The author links these facts to Sherrington's familiar experiments which shewed the great difficulty of obtaining anæsthesia by the section of any one root. He surmises that the stimulus overflowed to the other nerves still supplying the skin concerned. This is confirmed by the fact that section of two roots above, and two below the nerve experimented on, prevents the above phenomenon, the movements being then confined to the local group of muscles.

The importance of being aware of the above occurrence is pointed out, as many contradictory experiences are thus avoided. ERNEST JONES.

PSYCHOLOGY.

MATHEMATICAL PRODIGIES. FRANK D. MITCHELL, *Am. Journ.* (140) *of Psychol.*, Jan. 1907, p. 61.

IN a somewhat lengthy paper the author gives a summary of the chief particulars known regarding twenty-four mathematical prodigies (of whom only one is a woman) described in literature; an account of his own case; and some conclusions regarding mental calculation based upon the foregoing data.

His data do not enable him to speak of the influence of *heredity*. *Precocity* in calculation he finds to be normal. Skill in mental calculation is independent not only of *general education*, but of *mathematical ability* and education. Although mental arithmetic develops naturally out of *counting*, the fundamental operation is *multiplication*. Various mathematical and psychological *short-cuts* explain the speed attained by some of the prodigies in their mental operations. The distinction often made between *memory* and *calculation* is misleading; the process is always a true calculation. Many of the calculators hitherto supposed to belong to the visual *memory type* turn out to be of the auditory (or auditory motor) type, at least in calculation. At least two of the major prodigies, however, and possibly four, belong to the visual type. A convenient table facilitates comparison of the different cases described.

W. B. DRUMMOND.

THE PATHOLOGICAL ANATOMY AND PATHOLOGY OF
(141) **EPILEPSY.** JOHN TURNER, *Journ. of Ment. Sci.*, Jan. 1907.

THIS is an elaborate investigation into the microscopical anatomy of the brains of persons dying from epilepsy. Upon his observa-

tions the author bases his view that epilepsy is a disease occurring in persons with a defect of the nervous system, either congenital or involutional, and in whom also there is an abnormal state of the blood, characterised by a special tendency to intravascular clotting, and that the fits owe their exciting cause to sudden stasis of the blood stream, resulting from blocking of the cerebral vessels by these clots in some portion of the cortex.

(*a*) The evidence of intravascular thrombosis, which is the novel feature in the pathology of epilepsy advanced by the author, is found in the presence of large numbers of blood-plates forming clusters within the cortical vessels. When appropriately stained, they are found to be rich in phosphorus, a fact indicating their nucleo-proteid nature. These bodies often amalgamate, so as to form cylindrical masses, entirely filling up the lumen of a capillary or bigger blood-vessel. The *intra vitam* formation of these clots is shown by the fact that proximal to them the blood-vessel is frequently distended and ruptured, giving rise to ecchymoses of red corpuscles.

(*b*) Sclerosis and atrophy of small areas of the cerebral convolutions are frequently found, most commonly in the cornu ammonis, the cerebellum, and the parietal or occipital lobes. The author ascribes these atrophied areas to starvation and deprivation of their blood supply, arising from the thrombi which he finds so constantly present in the brains of epileptics.

(*c*) *Nerve Cells.*—The cortical nerve cells in epileptics show two types of change, the one congenital, the other degenerative. The former should be regarded as "stigmata," as they are found more especially in persons of under average intelligence, the changes being very clearly seen in the Betz cells of imbeciles. The latter, or degenerative changes, are similar to those induced by experimental ligature of the cerebral blood-vessels, and the author ascribes the condition to the interference with the cortical circulation arising from intravascular clotting. These changes consist of swelling of the nucleus, degeneration and death of the cell, and eventually its complete disappearance.

The author claims to have shown that among his cases 70 per cent. showed defective structure, and 90 per cent. showed, in the form of thrombi, a blood state capable of acting as an exciter of convulsions. The impaction of thrombi accounts also for the patchy degeneration of the nerve cells, as well as for the isolated areas of sclerosis and atrophy so common in the epileptic brain.

In an interesting addendum to the paper the author gives some observations upon the coagulability of the blood in epileptics. He found the difficulties considerable and the results by no means conclusive, but he formed the general opinion that, in most cases, there was, at or about the time of fits, a distinct acceleration of

coagulation. The mean coagulation time in 28 epileptics on 135 occasions was 120 seconds; in 13 cases of insanity on 38 occasions it was 136 seconds.

The bulk of the paper consists of a full and detailed account of the pathological appearances of the central nervous system of 41 cases of epilepsy. WM. ALDREN TURNER.

HYPERTROPHY, HYPERPLASIA, AND PSEUDO-HYPERTROPHY (142) OF THE BRAIN. (Hypertrophie, Hyperplasie, und Pseudo-hypertrophie des Gehirns.) MARBURG, *Arbeit a. d. neurol. Instit. a. d. Wien. Univ.*, Bd. 13, 1906, S. 288.

THIS paper is a contribution to the question of diffuse glioma of the brain, which sometimes leads to a considerable increase in its weight and size. In this condition the chief characteristic is a hyperplasia of the neuroglia, diffuse or circumscribed, with which, however, may be associated a true hypertrophy of the nerve cells and nerve fibres. To those cases in which softenings or other destructive processes of the nervous tissues have occurred, with a secondary overgrowth of the neuroglia, the author has applied the term "pseudo-hypertrophy."

True hypertrophy may be found unaccompanied by a hyperplasia, but this is very rare. It is not associated with any clinical symptoms, and frequently the intellectual development of the individual is good. Cases of marked hyperplasia, however, are usually met with in epileptic idiots.

The hyperplasia may be diffuse or circumscribed. In the former case numerous axis-cylinders and fibres are still preserved throughout the affected region, but in the latter condition they are met with only near the periphery.

With regard to the causes of these conditions, some authorities suggest that the hyperplasias may depend on the innate capacity of glia cells to proliferate; but the author considers that they are due to an embryonal or early infantile inflammatory process.

But when the hyperplasias are combined with a true hypertrophy of the nerve cells and fibres, the condition must have a different origin.

It is impossible to imagine that an increase of the hemispheres, it may be up to three times the normal, can take place in the adult. They must therefore be congenital, although a neoplasm, in the form of a circumscribed glioma, may develop later.

Some authorities have suggested that they may spring from a wandering germ cell which has retained its embryonal characters. This origin might explain the circumscribed tumours, but it could hardly be applied to the diffuse growths.

The author then discussed at some length the relations existing between growth of tissues and the secretions of the ductless glands. The influence of these may be indirect, *e.g.* through the disturbances of the blood circulation, as in the case of the suprarenal bodies, or direct, as in the case of the thyroid.

He referred to several cases which have been described by other observers in which brain hypertrophy, hyperplasia, or other mal-formations have been present in cases which showed some alterations in the thyroid, thymus, hypophysis, epiphysis, or suprarenal bodies. Generally too, in these cases, more than one of these glands were affected. But whether the hypertrophy and hyperplasia are to be attributed to a deficiency or excess of secretion is not known.

R. G. Rows.

(143) **THE NORMAL AND PATHOLOGICAL ANATOMY OF THE POSTERIOR SPINAL ROOTS. (Studien zur normalen und pathologischen Anatomie der hinteren Rückenmarkswurzeln.)** Levi, *Arbeiten a. d. neurol. Instit. a. d. Wien. Universität*, Bd. 13, 1906.

The author's research is confined to that part of the posterior root where it joins the cord. In all segments there is a glial layer which envelopes the roots and sends processes in between the fibres, forming a thick network on joining the connective tissue. This junction of the glial and connective tissue elements of the root is found in the cervical region, situated inside the cord, and in the lumbar and sacral region outside; but in the dorsal region it takes place at the cord margin. The diminution in calibre which takes place where the root pierces the pia arachnoid coincides only in the dorsal region with the above-mentioned point.

Where Schwann's sheath is lost and the glia begins, one finds that apparently the medullary sheath is wanting too, giving rise to the clear ring seen by the Weigert method. This clear ring indicates where the peripheral nerve becomes part of the central nervous system, and in the dorsal and even in the cervical region may correspond in situation to that point where the root undergoes a constriction in passing through the pia. This, however, is not the case in the lumbar and sacral cord.

The changes in early Tabes are seen to commence where the medullary sheath loses its staining reaction, *i.e.* in the lumbar and sacral cord outside the constriction, so that this loses its alleged significance in the pathology of the disease.

David Orr.

THE TISSUE CHANGES IN THE BRAIN OF SYPHILITIC INFANTS. (Über Gewebsveränderungen im Gehirn luetischer Neugeborener.) RANKE, *Neurol. Centralbl.*, Nos. 3 and 4, 1907. (144)

FIFTY brains were examined at varying periods of development, ranging from the third month of fœtal life to some months after birth.

The author found proliferative changes in the capillaries, and in the intima of the arteries and veins, some of the latter being filled with endothelial cells. In many instances plasma cells infiltrated the adventitia; and although passing beyond the limits of the lymph sheath, the infiltration was restricted to the tissues around the vessel. The neuroglia showed proliferative changes. In addition to diffuse lesions there were isolated foci, in which were recognisable plasma and mast-cells, elongated adventitial elements, and neuroglia hypertrophy. When these were present in the cortex, they were associated with nerve cell degeneration. In three cases spongioblasts were found.

The pia was opaque and infiltrated by plasma cells, fibroblasts, and large round cells.

The above pathological changes are the result of persistence of normal proliferation, in consequence of syphilitic irritation.

In two cases the author found spirochoetæ in large numbers amongst the endothelial cells of the vessels, in the muscular coat, in the adventitia, and even in the brain substance.

DAVID ORR.

THE CHANGES FOUND IN THE CENTRAL NERVOUS SYSTEM IN PUERPERAL ECLAMPSIA. (Die Befunde am Zentralnervensystem bei der puerperalen Eklampsie.) POLLAK, *Arbeiten a. d. neurol. Instit. a. d. Wien. Universität*, Bd. 13, 1906. (145)

IN the ten cases investigated, the anterior cornual cells, the nuclei of motor cranial nerves, the cells of Clarke's column, and those of the spinal ganglia all showed more or less severe changes affecting both chromophile elements and the nucleus. The ganglion cells of the posterior horns showed much less alteration than those of the motor cells. The glia cells also showed alterations.

In the cerebral cortex the large pyramidal cells were affected, the chromophile material being collected at the cell base, and in many there was evidence of phagocytic action.

The degenerative changes in the cord did not show a like intensity in all areas, but specially affected the motor cells.

DAVID ORR.

RESEARCHES ON THE CHANGES OF THE NEUROFIBRILS
(146) **AFTER VARIOUS DISTURBANCES OF NUTRITION.** (**Recherches sur les changements des neurofibrilles consécutifs aux differents troubles de nutrition.**) MARINESCO, *Le Nevraxe*, Vol. viii., Fasc. 2-3.

THE author refers to the work of Cajal and Tello, who found that the neurofibrils undergo a considerable hypertrophy in animals exposed to cold, and who have suggested that this was a morphological change due to the cold. This hypertrophy was also seen by them in cases of hydrophobia, and was attributed to altered function.

Marinesco considers that they all result from disturbances of nutrition.

Changes in the chromophile substance often advance parallel to the affection of the neurofibrils; but sometimes the chromophile substance may be considerably altered, while the neurofibrils remain intact. This is well seen in the hibernating hedgehog, in which, although the chromophile elements have largely disappeared, the neurofibrils are arranged regularly and are not enlarged.

The author, as an explanation of this difference in results, suggests that it may be due to the fact that Cajal and Tello examined the cells of cold-blooded animals, while he employed warm-blooded animals, in which a more active nutrition goes on during hibernation.

In his experiments he has found that hypertrophy and coalescence of the fibrils does occur in young dogs and cats if they are exposed to a temperature of 10°-15°, but exposure to temperatures below or above this (2° or 30°) leaves them almost intact. It is to be noted also that if a marmot, which falls into a hibernating sleep at 6°, is exposed to a temperature of 0°, it awakens, and begins to shiver and produce heat. It is possible that the tumefaction of the fibrils at low temperatures may be a means of defence against a dissipation of energy.

It is not, however, a necessary result of low temperatures, or rather, it only occurs under special conditions.

Further experiments have proved that hypertrophy and coalescence of the neurofibrils are produced by various other disturbances of nutrition, and especially by intoxications, *e.g.* hydrophobia, morphia, strychnine, and moreover, a combination of these poisons with inanition or exposure to cold produces a much more marked alteration.

R. G. Rows.

NERVE DEGENERATION. (La nature intime du processus de (147) Dégénérescence des Nerfs.) G. MARINESCO, *La Presse médicale*, Feb. 16, 1907, p. 105.

AFTER remarking that almost nothing is known of the intimate nature of nerve degeneration, Marinesco reviews briefly our knowledge of the morphological and chemical changes which occur in the process of Wallerian degeneration. He believes that both the axolysis and myeline changes are the result of a species of digestion by means of ferments secreted by the nuclei of Schwann's sheath. He gives a general account of some nerve transplantation experiments in support of this view. When a piece of nerve was transplanted under the skin there were some irregularly oriented apotrophic cells (cells produced by proliferation of the cells of Schwann's sheath, which have a guiding influence on nerve fibres of new formation) but no new fibre formation. As the result of Auto- and of Homo-transplantation there was always new fibre formation and appearance of apotrophic guiding cells. These cells are the essential feature of both degeneration and regeneration. In cases of Hetero-transplantation there was no real Wallerian degeneration, but rather a necrosis. No apotrophic cells were formed. The piece of transplanted nerve apparently provokes the formation of antibodies which are cytotoxic to it—hence there are no apotrophic cells formed, and consequently no degeneration or regeneration is possible.

J. H. HARVEY PIRIE.

CLINICAL NEUROLOGY.

PSEUDO-HYPERTROPHIC MUSCULAR ATROPHY. CHARLES (148) INGBERT, *Journ. of Nerv. and Ment. Dis.*, Jan. 1907, p. 2.

AFTER dealing with the classification of muscular atrophies, this paper describes a fairly typical case of pseudo-hypertrophic paralysis in a lad who died of typhoid fever at the age of 20.

The pathological examination of the spinal cord showed nothing abnormal except some chromatolysis of the nerve cells of the anterior horn, which the author attributes to the typhoid infection, and a diminution in the number of nerve cells in the cord below the fourth lumbar segment.

A careful investigation of the nerve roots shows that there is a relative diminution of the size of the motor as compared with the sensory roots.

In the left fifth lumbar motor root only 5171 nerve fibres were counted as compared with 10,366 in a corresponding normal nerve

root; on the other hand, the normal root contains 4800 nerve fibres per square m.m., whilst the abnormal contains 4040. It is therefore evident that the root investigated has a smaller area, contains a smaller number of fibres, and has a smaller number of fibres per square mm., that is to say, there is a greater diminution of the number of small fibres of the roots as compared with the large fibres. The muscles showed the usual changes.

The pathological findings add support to the conclusion of other investigators that pseudo-hypertrophic muscular atrophy is primarily a disease of the muscles, and that the changes in the spinal cord and in the spinal nerve roots are secondary.

F. E. BATTEN.

(149) **MYOPATHY OF THE DISTAL TYPE AND ITS RELATION TO THE NEURAL FORM OF MUSCULAR ATROPHY (CHARCOT-MARIE TOOTH TYPE).** WILLIAM G. SPILLER, *Journ. of Nerv. and Ment. Dis.*, Jan. 1907, p. 14.

THIS paper gives a good description of a case of progressive neuritic muscular atrophy, and compares it with a case of muscular atrophy having many features of the above type, but differing in that the proximal muscles were affected and contraction occurred. The pathological examination of the spinal cord in the second case showed no change.

The author starts with the statement that, when first described, primary neuritic muscular atrophy was considered to be so sharply defined from other types of muscular atrophy that confusion could not occur. After detailing the characteristic features of the type, he seems to lay special stress on the almost constant absence of all contractions of tendons, and would hesitate to accept many cases described as progressive neuritic muscular atrophy because contractions were present. On such grounds it would be possible to refuse to accept the case here described, because there was no evidence that the disease had affected other members of the family, or had been transmitted from a previous generation.

The fact whether contraction of tendons occurs, depends upon the relative affection of the antagonistic muscles, and although in most cases of neuritic muscular atrophy no contractions occur, yet to exclude a case from the group because contraction is present seems to me irrational.

The author then gives a most excellent digest of atypical cases of neuritic muscular atrophy, and shows that many cases exhibit features which bring them into close association with other recognised types of myopathy.

The paper concludes with the description of a case in which Dr Spiller hesitated to make the diagnosis of neuritic muscular

atrophy, because the upper arm, thigh, and trunk muscles were much atrophied. Contractions were also present in the lower extremities. Section of the cervical and lumbar cord showed no pathological change, but the muscles showed long-standing and pronounced degeneration, F. E. BATTEN.

MYASTHENIA GRAVIS. C. EUGENE RIGGS, *Journ. Amer. Med. Ass.*, (150) Dec. 15, 1906, p. 1969.

IN each of the three cases here recorded ophthalmoplegia was present in addition to the general muscular weakness. All three patients were males and their ages were 37, 35, and 45 respectively. In the first there was no apparent cause, in the second the symptoms came on shortly after an attack of influenza, and in the third a period of great anxiety and overwork preceded the onset. They all exhibited ptosis and the nasal smile, and the myasthenic reaction—the rapid exhaustion of the affected muscles by faradic stimulation—was obtained in each case. In the first, the diplopia was followed by myasthenia in less than a week; in the second, two months elapsed between the onset of the diplopia and the first appearance of myasthenic symptoms; in the third, transient symptoms preceded the real inception of the disease by four months. One of the patients died after a year's illness, and another was practically *in statu quo* a year after discharge from hospital. The third had been lost sight of but had probably since died. In discussing the differential diagnosis, the writer emphasises the difficulty of distinguishing between myasthenia gravis and hysteria in the early stages. As regards the pathology, there is probably circulating in the blood a toxin—perhaps the product of an enlarged or modified thymus, or an abnormal lymphoid tissue infiltration throughout the body—affecting the lower motor neuron. Strychnine, gentle massage, and galvanism were found to be the most efficient agents in treatment.

HENRY J. DUNBAR.

CHILDBIRTH IN ADVANCED TABES. (Eine Geburt bei vorgeschrittener Tabes dorsalis.) (151) ZACHARIAS, *Muench. med. Wochenschr.*, Feb. 12, 1907.

COMPLETE details are given of a case in which a multipara aged 43, who had suffered for nine years from tabes, was delivered painlessly of a child. The unusual points were that the abdominal muscles did not come into action till the last four pains, when the head was being extruded; that there was absolutely no suffering, and that a rupture of the uterus appeared to be imminent towards the end of the labour.

The tabes was well advanced, the chief symptoms being immobile pupils, complete loss of superficial and deep reflexes in arms and legs, lightning pains, anæsthesia on the legs, diminution of touch and temperature sense on legs and ulnar side of arms, inco-ordination, and interference with visceral functions.

JOHN D. COMRIE.

CASE OF IDIOPATHIC TREMOR SIMULATING IN PART THE
(152) **PICTURE OF DISSEMINATE SCLEROSIS. (Intorno ad un caso di tremore essenziale simulante in parte il quadro della sclerosi multipla.)** IGINIO BERGAMASCO, *Rivist. di Pat. nerv. e ment.*, Jan. 1907, p. 4.

THE patient was a man of 64. He was admitted on account of an aortic aneurism, which killed him a few days later. On examination of the nervous system, three abnormal physical signs were detected:—(1) Marked intention tremor in both arms; it was absent at rest, and increased on reaching toward an object; the oscillations were wide and slow, and affected first the fingers, then hands. There was no tremor of the lips, tongue, head, legs, or trunk. (2) Nystagmus, which occurred readily apart from any forced movement of the eye. (3) Speech slow and scanning. The first symptom had been present for fifteen years, the other two had been noticed for only eighteen months.

At the autopsy the central nervous system was perfectly normal, even on microscopic examination, except for diffuse arterial disease and a recent (week old) thrombotic softening, the size of a bean, in the left cerebellar hemisphere.

The writer points out the difficulty in diagnosis, and discusses the various causes of similar tremors. There was no other case in the family.

ERNEST JONES.

TUMOUR OF THE SPINAL CORD, LEADING TO DESTRUCTION
(153) **OF THE LUMBAR REGION, HYDROCEPHALUS, DOUBLE OPTIC NEURITIS, AND PAINLESS LABOUR.** TAYLOR, *Boston Med. and Surg. Journ.*, Feb. 7, 1907.

THE paper gives in detail the symptoms presented by a woman, aged 24, who presented some unique features. The complaint began suddenly, with pains down the legs and in the back, which in the course of three months became extremely severe, and were accompanied by increasing weakness, resulting in almost complete paraplegia within this time. Up to this time, as she was pregnant, the symptoms were attributed to this condition; but later she developed headache, mental aberration, complete anæsthesia below

the waist, paraplegia, and bladder trouble. Nearly four months after the onset of the symptoms, the patient gave birth to a healthy child without pain, and without the use of the abdominal muscles. The points of peculiarity in the labour were its painless character, the absence of any instinctive use of the abdominal muscles, and the firm contraction of the uterus after labour, so that no hæmorrhage occurred. There were no subsequent obstetrical complications. The patient died six months after the onset of symptoms. Post-mortem there was found an extensive tumour of the spinal cord, destroying the lumbar region and associated with internal hydrocephalus. The occurrence of optic neuritis—quite unusual in tumours of the cord—is explained by the writer as due to the hydrocephalus induced by the wide spread of the tumour. JOHN D. COMRIE.

MENINGITIS IN MUMPS. (Contribution à l'étude de la méningite (154) ourlienne.) FELICIANO, *Thèse de Paris*, 1907.

MENINGITIS in mumps may occur in children or adults, but in the latter, almost exclusively in men and in association with orchitis. Like that which develops in typhoid fever, scarlatina, and other infectious diseases, meningitis complicating mumps belongs to the serous variety. The symptoms to which Dupré gave the name of meningism, correspond in most cases, as cyto-diagnosis proves, to a real inflammation of the meninges. "Ever since lumbar puncture has entered into current practice, meningism has lost ground, and its place has been taken by serous meningitis" (Hutinel). The brain lesions are usually ill-marked in serous meningitis, but small extravasations of blood may occur, and even softening of the brain substance, as is manifested clinically by hemiplegia and aphasia.

Three forms of meningitis in mumps are described. The first is an incomplete one in which the symptoms are so slight that a clinical examination must be supplemented by lumbar puncture to establish the diagnosis. Headache, pyrexia, and a slow pulse may be the only symptoms. The second form is one with complete symptomatology, but favourable evolution. Headache, vomiting, stiffness of the neck, and Kernig's sign are present. Occasionally ocular paralysis and delirium may develop. In two or three days the symptoms disappear. In very rare cases aphasia, hemiplegia, or agraphia with difficulty of speech and uncertainty of gait may arise and persist. The third is a grave form. The onset is the same as in the second, but the disease runs a rapidly fatal course.

The fluid obtained by lumbar puncture is usually clear. In most cases there is a pure lymphocytosis.

The prognosis is favourable as a rule. In most cases the meningitis remains serous, and the symptoms disappear fairly rapidly. The possibility of hemiplegia and aphasia must be considered, but such complications are very rare.

Treatment consists in the employment of hot baths several times daily. Lumbar puncture will often afford temporary relief. Leeches behind the ears, wet-cupping of the spine, cold applications to the head, and such drugs as calomel, potassium bromide, and chloral, may also be employed with advantage.

J. D. ROLLESTON.

(155) **ON THE PRESENCE OF THE MENINGOCOCCUS AND THE MICROCOCCUS CATARRHALIS IN THE NASAL CAVITIES, AND DISINFECTION WITH PYOCYANASE. (Ueber das Vorkommen des Meningococcus und des Micrococcus Catarrhalis im Nasenrachenraum und Desinfectionsversuche mit Pyocyanase bei diesen Infectionen.)** JEHLE, *Wien. klin. Wochenschr.*, Jan. 3, 1907.

THE writer states that this substance has been used as a germicide in anthrax, cholera, diphtheria, and typhoid fever. It is supposed to contain a bacteriolytic enzyme, and was used by him both against culture of the bacteria in question and in cases of natural infection. The method of experimentation was to add drops of the pyocyanase to one cubic centimetre of cerebro-spinal fluid or of normal salt solution containing organisms. After the lapse of various periods of time, attempts were made to produce cultures of the bacteria, generally in vain.

With regard to patients suffering from influenzal attacks and having micrococcus catarrhalis in the nasal secretion, he found that after one application no more organisms could be obtained from the nose when 48 hours had elapsed. With regard to 39 cases of patients suffering from and contacts with epidemic meningitis in whose nasal cavity the meningococcus was discovered, two applications at most were required to effect the disappearance of the organism.

On the other hand the writer states that pyocyanase had little or no effect upon the streptococci, staphylococci, pneumococci, bacillus coli, and Friedländer's bacillus, commonly found in the nose.

JOHN D. COMRIE.

A CASE OF CEREBRO-SPINAL MENINGITIS, WITH OBSERVA-
(156) TIONS ON A PLEOMORPHIC DIPLOCOCCUS OBTAINED BY LUMBAR PUNCTURE. DARLING and WILSON, *Brit. Med. Journ.*, Feb. 23, 1907.

THIS paper records a fatal case of sporadic cerebro-spinal meningitis, with the bacteriological findings in the spinal fluid.

The organism present was a gram-positive coccus, which assumed bacillary form on Conradi-Drigalski medium; did not liquefy gelatine; clotted milk; produced no indol; produced no gas in sugar-containing media; and was feebly pathogenic for rabbits. The writers believe it to be identical with the micrococcus rheumaticus. They make some general observations upon the assumption by other cocci of the bacillary form, and give a photo-micrograph of the present organism in this state.

JOHN D. COMRIE.

CELLS OF THE EXUDATE IN EPIDEMIC CEREBRO-SPINAL
(157) MENINGITIS. (Cellules de l'exudat dans la méningite cérébro-spinal épidémique.) SPERONI, *Presse méd.*, Feb. 6, 1907.

THE writer gives the result of his examination of the cells present in eleven acute cases of this disease examined post-mortem. He found both in these, and in other acute cases examined by lumbar puncture, chiefly polynuclear leucocytes, many of which contained 2 to 30 diplococci, in smaller number lymphocytes and large mononucleated leucocytes, always more or less red-blood corpuscles, and finally fibroblasts. In more chronic cases of three to eight months' duration, the lymphocytes and large mononuclear cells predominated; the polynuclears were few in number.

JOHN D. COMRIE.

CASES OF MENINGITIS, WITH ISOLATION OF DIPHTHERIA
(158) BACILLUS FROM CEREBRO-SPINAL FLUID. MORRELL and WOLF, *Journ. Am. Med. Ass.*, Dec. 29, 1906.

THIS is record of a case in which a coloured infant died with symptoms of meningitis. Both on lumbar puncture and after death, organisms showing the various characters of the *B. Diphtheriæ* were isolated by culture. The child after death showed various tuberculous lesions also. The writers state that they have been able only to find one previous case in which this bacillus was obtained from the central nervous system.

JOHN D. COMRIE.

A CASE OF TRAUMATIC "LATE" APOPLEXY. (Ein Fall von traumatischer Spätapoplexie.) HOCHHEIM, *Muench. med. Wochenschr.*, Jan. 29, 1907.

(159)

THE writer records the case of a workman, aged 34, free from Bright's disease or heart-disease, with healthy arteries and no history of syphilis, who nine weeks after a severe fall upon the back of the head developed a left-sided hemiplegia. Immediately after the accident he had to be helped to his feet and complained of great pain, but was able to resume his work. After some weeks he became forgetful and indifferent at work, and at the end of nine weeks one day was completely confused. The next night he became completely stuperose, the left face, arm, and especially leg were paralysed, and the sphincters relaxed. Within seven months recovery of mental power, control of the sphincters, and partial return of the use of hand and leg were established.

The writer gives references to similar cases, and mentions two theories accounting for the connection of trauma with the apoplexy: that of Bollinger, that the injury causes a softening and necrosis; and that of Langerhans, that the cerebral vessels are damaged and weakened.

JOHN D. COMRIE.

SYMPTOMATOLOGY OF CEREBRAL HEMIPLEGIA. (Zur Phänomenologie der cerebralen Hemiplegie.) Z. BYCHOWSKI, *Neurolog. Centralbl.*, 1907, p. 155.

(160)

BYCHOWSKI describes the phenomenon which has already been independently recorded by Grasset and Gaussel (*Revue Neurologique*, 1905, p. 881). It consists in the fact that in organic hemiplegia, whilst the patient can raise either lower limb separately from the bed in the supine posture (the hemiplegic limb being less powerfully raised than the sound one), it is usually impossible for him to raise both lower limbs simultaneously. Moreover, if the hemiplegic limb be raised alone, and we then passively lift the sound limb, the hemiplegic limb at once falls down. But whereas the French observers ascribe the phenomenon to the inability to fix the pelvis by the muscles of the hemiplegic side, Bychowski's explanation is much less clear. So far as can be gathered from a somewhat involved statement, he ascribes the difficulty of raising both lower limbs to the fact that each cerebral hemisphere is accustomed to the performance of alternate rather than of simultaneous movements of the lower limbs; hence when one hemisphere is paralysed, the other hemisphere in the process of compensation succeeds better with alternate movements than with simultaneous ones.

PURVES STEWART.

TWO CASES OF ENDOTHELIAL SARCOMA OF THE BRAIN—(161) SUCCESSFUL REMOVALS WITH SUBSEQUENT DEATH.

C. EUGENE RIGGS, *St Paul Med. Journ.*, Jan. 1907.

IN both cases the tumour was situated in the posterior part of the second left frontal convolution and the adjacent motor area, giving rise to headache, epilepsy, aphasia, optic neuritis, turning of the head and eyes to the opposite side, and mental impairment. A full account of each case is given, and then the author discusses the question of palliative operation in cases of brain tumour with regard to which his conclusions are as follows: 1. Palliative operations should always be done early and before optic atrophy has occurred. 2. Pain may kill. Headache, therefore, may be of such intensity as to demand this procedure. 3. Cerebral decompression should be regarded as a definite, recognised means of surgical endeavour in every inoperable case.

HENRY J. DUNBAR.

CASES ALLIED TO AMAUROTIC FAMILY IDIOCY, WITH (162) REMARKS ON THE PATHOGENESIS OF THE AFFECTION.

ALFRED GORDON (Philadelphia), *New York Med. Journ.*, Feb. 16, 1907.

THE two cases brought forward by the author may be placed, he contends, in the category of cases of amaurotic family idiocy, owing to the following characteristics—blindness commencing very early in life, mental deficiency from infancy, optic atrophy, and the fact that the two patients (brother and sister) were the offspring of Russian Hebrews.

Briefly stated, the two cases were as follows:—

Case 1.—Male æt. nine years. Walked at nine months, spoke at two years.

When he was twelve months old the mother noticed he could not see well. He has always been very backward and childish.

At the age of three years he began to grow very stout, and this stoutness has constantly increased, although he eats very little.

Thyroid gland impalpable, reproductive organs infantile, reflexes normal, slight instability of head.

Patches of absorption of choroidal pigment in both eyes, with partial atrophy of optic nerves.

Six fingers on each hand, and six toes on each foot.

Case 2.—Female, æt. thirteen years.

Began to talk and walk at age of three years, and at same time

blindness was noticed by parents. Very backward, foolish, and apathetic. Extremely stout, and gradually becoming more so. Thyroid gland enlarged. Optic atrophy.

These cases the author considers very suggestive in view of the possibility "that some profound anomalies in the structure and function of the ductless glands are the real cause of the diffuse anomalous condition of the cells of the nervous system." Both the recorded cases showed changes in the thyroid gland; from the illustration of the boy, his general appearance is seen to be markedly cretinoid; and rather than reflexion on the possible relation between thyroid secretion and amaurotic family idiocy, one is forced to consider the propriety of classing these cases under that term. A. DINGWALL-FORDYCE.

A DISCUSSION OF SOME IMMEDIATE AND SOME REMOTE
(163) **CONSEQUENCES OF CRANIAL INJURIES, BASED ON THREE CLINICAL HISTORIES WHICH ILLUSTRATE THE EXTRADURAL, SUBCORTICAL, AND INTERMENINGEAL TYPES OF INTRACRANIAL HEMORRHAGE.** H. CUSHING (of Baltimore), *N. Y. Med. Journ.*, Jan. 19, 26, Feb. 2, 1907.

THE *first* patient sustained a meridional fracture of the skull by a crushing force applied laterally. Extradural hæmorrhage followed from rupture of the meningeal artery, and the patient exhibited compression symptoms, without preceding symptoms of concussion. A somewhat misdirected operation was followed by motor aphasia, which eventually cleared up almost completely. About three years later a pulsating exophthalmus developed, which was affected little or none at all by ligation of the internal carotid.

In the comments upon this case the author refers to the extreme importance of the examination of the fundus of the eye in the determination of acute intra-cranial tension. With practice slight grades of venous stasis and early œdema should be recognised in the disc. The side upon which the pressure is being exerted can be readily determined by this means. In spite of the critical injury and its subsequent complications, the patient made good progress, and remained free from the most common sequelæ of cranial injuries, *i.e.* "post-traumatic neuroses."

The *second* patient was the victim of a penetrating stab wound of the skull. The left side of the brain was punctured directly behind the middle of the Rolandic fissure; subcortical, post-central hæmorrhage followed with contra-lateral sensory disturbances and visual word-blindness. An operation for the evacuation of the clot was followed by a rapid disappearance of

all the symptoms except astereognosis. Faradisation of the motor cortex was performed.

The author has been led to the opinion by this case and a series of others, that "a sufficient lesion of the hemisphere posterior to the sulcus centralis (Rolando), with no precentral lesion whatever, may produce motor symptoms which, however, are unaccompanied by degeneration of the pyramidal tract, and hence should not be regarded as a motor paralysis in the common acceptance of the term."

The *third* case showed an old penetrating bullet wound of the skull, which involved the left frontal lobe. The patient suffered from epilepsy with vocal aura. An operation was followed by improvement, but not by recovery.

In traumatic epilepsy of long standing, the author believes, that in the majority of cases the seizures are greatly lessened in intensity, and that in a small percentage they do not recur at all after operation. The gradual lessening in the severity and number of attacks is a more favourable sign than the immediate post-operative cessation.

Concerning the psychic disturbances which follow a fracture of the skull, Brun has stated that in a study of 470 cases he found subsequent psychic disturbances in 34 per cent.; few of these, however, showed permanent alterations in mentality. English believes that some degree of mental impairment follows fracture in 10 per cent. of cases. Meyer estimates that these cases represent about 1 per cent. of asylum inmates. C. H. HOLMES.

SIXTH NERVE PARALYSIS, AND THE CEREBRO-SPINAL PATH
(164) **OF INFECTION. (Les paralysies du moteur oculaire externe et la voie céphalorachidienne.)** FERNAND LÉVY and ALPHONSE BAUDOUIN, *Rev. Neurol.*, Feb. 15, 1907, p. 102.

THE question of 6th nerve palsy following the introduction of a toxin into the cerebro-spinal canal is still quite recent. It was first observed by certain German surgeons following operations in which anæsthesia had been induced by the injection of stovaine into the lumbar part of the spinal canal. Two cases were reported in which diplopia followed this procedure; both recovered spontaneously, in the first case after six months, in the second at the end of a fortnight. In both cases about a fortnight elapsed after the operation before any signs of diplopia were noted. Roeder suggested, as an explanation, that the paralysis was due to the toxine of stovaine circulating along the subarachnoid space.

The authors report two further cases following the deep injection of a mixture of alcohol and cocaine for trigeminal neuralgia.

In the first case, that of a woman aged 50, two injections were made into the superior maxillary division, in the pterygo-maxillary fossa, without any ill effect. The inferior maxillary division was then injected at the foramen ovale. This was done twice, and immediately following the second injection a paralysis of the 6th nerve on the same side occurred. The condition still persisted at the end of a month, though it had improved somewhat. The second case was similar in all respects to the first, but in addition to the 6th nerve palsy following the second injection, a slight degree of ptosis was noted also. On the eighth day the diplopia had disappeared.

The authors discard any idea of the cocaine solution spreading into the orbit and causing paralysis in this manner, as the injection of the superior maxillary division was followed by no bad results. In their opinion some of the solution passed through the foramen lacerum medium and infected the subarachnoid space through which the 6th nerve passes, before entering the wall of the cavernous sinus. If this explanation is correct, the authors think that the paralysis is peripheral rather than central. The reason why the 6th nerve suffers in all these cases is not clear. It appears especially susceptible. The cases suggest the possibility of a similar path of infection in those transitory ocular palsies which are met with in acute and tubercular meningitis, as well as in secondary syphilis. C. M. HINDS HOWELL.

TRANSIENT OPHTHALMOPLEGIA EXTERNA ASSOCIATED
(165) **WITH ATTACKS OF SEVERE HEADACHE.** J. HERBERT FISHER, *Ophthal. Rev.*, Feb. 1907, p. 31.

UNDER this heading the author gives a full account of five cases which he has personally observed. He regards them as being probably allied to the cases recorded by Ormerod and Spicer in Vol. xvi. of the *Transactions of the Ophthalmological Society*, under the title of "Recurrent Paralysis of Ocular Nerves." The author does not think the term ophthalmoplegic migraine should be applied to these cases for two reasons: first, that the headache is not exactly of the type which is usually found in migraine; and secondly, because the term tends to detract from a search for an organic lesion, which has been discovered in some of the cases, and is, in the author's opinion, probably present in them all. The pain is not necessarily unilateral, and the position of maximum intensity is variable—being occipital in two of the cases—retro-ocular, temporo-frontal, occipital and retro-ocular in the others.

The opportunities for complete examination in such cases as the author describes have been few, but Gübler has described a

plastic exudation in the basal subarachnoid space in one case, in which fibrous adhesions were also present around the origin of the third nerve. In other cases a fibroma or chondro-fibroma involving the ocular nerves has been described. No nuclear lesions have ever been noted. C. M. HINDS HOWELL.

A CLINICAL AND ANATOMO-PATHOLOGICAL OBSERVATION OF THE EYES OF A CASE OF GENERAL PARALYSIS. (166) **(Observation clinique et anatomo-pathologique des yeux d'une paralytique générale.)** A. RODIET and BRICKA (of Marseilles), *L'Encéphale*, Sept.-Oct. 1906.

THE clinical examination of the patient, aged 35, showed sluggish reaction of both pupils to light; the left pupil was wider than the right, and showed only faint reaction to accommodation. Ophthalmoscopic examination showed a slight degree of optic atrophy. The histological examination disclosed an optic neuritis due to the proliferation of the connective tissue elements and the neuroglia between the bundles and nerve fibres of the optic nerve. The nerve cells in the retina showed inflammatory and destructive changes. The case is insufficiently reported.

C. MACFIE CAMPBELL.

PARALYTIC PTOSIS IN HYSTERIA. **(Le ptosis paralytique dans l'hystérie.)** (167) CH. SAUVINEAU, *Rev. Neurol.*, Feb. 15, 1907, p. 97.

THE author records two unusual examples of hysterical paralysis.

The first case was that of a girl aged 11½ whose mother gave the following history. The girl had been in her ordinary health, which was good, till a month previously. At this time she had an attack in church, felt sick, was pale, fainted for a short time, and afterwards slept for about an hour.

Ten days previously awoke with complete bilateral ptosis. On examination the palpebral fissure on either side was reduced to about 3 mm. In order to see, the child held her head back. There was no overaction of frontales, or orbicularis to assist the paralysed levator palpebræ. There was no spasm of orbicularis. In addition, the child had a very well marked left hemianæsthesia, but without any limitation of the visual field.

The diagnosis of hysteria was made, and a rapid cure effected by faradisation and suggestion. A relapse followed in a fortnight, but on this occasion suggestion alone sufficed to effect a cure, which has remained permanent.

The second case was that of a girl aged 13.

Two years previously she had suffered from phlectenular ulceration of the cornea, which had produced an intense photophobia—the condition relapsed from time to time. With the photophobia, blepharospasm developed, at first associated only with the presence of corneal ulceration, later becoming continuous. On examination the patient presents the following appearance:—

On the left side there is a well marked condition of blepharospasm, which is accentuated when the child is told to open her eye, or when an attempt is made to do so. On the right side there is a partial condition of ptosis, unassociated with any spasm. There is marked overaction of the frontales, with elevation of the eyebrow. The head is carried thrown back, in order that the patient may see.

There is no loss of sensation of any kind, and the child's general health is excellent. The application of sparks from a high frequency apparatus was ordered, combined with statical electricity.

As a result of this, during the first sitting, the ptosis cleared up, but the blepharospasm, on the other hand, made but slight improvement, but gradually passed off during the next fortnight. The eyes were then examined for errors of refraction, and a compound hypermetropic astigmatism discovered. Glasses to correct this were ordered and so long as the child wears them, all goes well. If, however, they are removed, fibrillary tremors at once commence in both eyelids, at the same time the eyes oscillate quickly, the child never looking in any definite direction, or fixing any particular point.

In conclusion, the author defines the diagnosis between ptosis and blepharospasm, laying special stress on the implication of the lower lid in the latter condition. He draws attention to errors of refraction as being a frequent source of tic and blepharospasm, and emphasizes the necessity of carefully testing for such errors, as they may otherwise escape attention.

C. M. HINDS HOWELL.

DISTURBANCES OF THE URINARY SYSTEM IN HYSTERIA.

(168) **(Über Störungen im Bereiche des Harnapparates bei Hysterie.)** ALFRED HOCK, *Prag. Med. Woch.*, Feb. 7 and 14, 1907. S. 65 u. 79.

THE author has shortly put together an account of the more common manifestations of hysteria that are seen in connection with the urinary apparatus. He emphasises the importance of their study and gives a series of instances in which serious operations had ignorantly been undertaken for their relief.

1. He mentions first hysterical renal colic, which by its

intense pain radiating along the ureter to the groin, severe strangury with suppression of urine, hæmaturia, vomiting, and pyrexia gives rise to the greatest difficulty in diagnosis; normal kidneys have frequently been incised and even removed through the error thus brought about. Two new cases are briefly described, one in a woman of 26, the other in a boy of 13. The occurrence of hæmaturia in hysteria has of course been much doubted, but recent exact observations have shewn that this symptom may undoubtedly occur. Hock mentions several other causes of hæmaturia proceeding from a healthy kidney.

2. Hysterial nephralgia alone is usually attributed to mobile kidney, which explains the want of success that operations undertaken for this condition shew.

3. The anomalies of urinary secretion in hysteria are two—excess and diminution. The polyuria is usually transient, taking place at the end of a hysterical attack; only the watery constituent is increased. Permanent polyuria occurs chiefly in male hysterics; the commonest time is between the ages of 25 and 50; it may begin suddenly or gradually; the amount passed may exceed 40 or 50 pints a day; tenesmus is common in the condition, and retention, necessitating catheterisation, may occur; the chlorides are in excess, the other constituents not; in severe cases when the polydipsia does not equal the polyuria advanced wasting may ensue.

4. Hysteric anuria is not so firmly established as a true undoubted phenomenon, in spite of Charcot's teaching. Gilles de la Tourette distinguished between *transitory* complete anuria and incomplete oliguria, and *permanent* anuria in which vicarious secretion takes place from the stomach, bowels, skin, and salivary glands.

5. The commonest bladder manifestation is cystalgia, usually diagnosed as irritable bladder. The urine is normal, but the mucous membrane is so sensitive that distension cannot be allowed and pollakuria is usually marked. Sphincter spasm may prevent the introduction of instruments.

6. Hysteric retention is the best known symptom, though it is very rare. The author mentions a case in a man who had been catheterised daily for eighteen months. He was temporarily cured by suggestion but was admitted later into a strange hospital for retention, and ultimately died of an ascending infective condition. The author holds that the non-development of trabeculæ in the bladder (*Balkenblase*) differentiates the state from retention due to organic nervous diseases, the commonest of which in this connection is tabes. Hysteric retention is due in some cases to paralysis of the detrusor, with or without anæsthesia of the mucous membrane, in others to spasm of the sphincter. De la Tourette

related a case of the latter variety in which the impassability of the sphincter led to the performance of urethrotomy.

7. Hysteric incontinence is not so common, but marked cases are on record. The author records two, one in a woman of 23, the other in a woman of 20; in both cases there was complete anæsthesia of mucous membrane, and ignorance as to the act of micturition, until the skin was felt to be wet. In the former instance the origin of the condition dated to a New Year's night when the patient—a servant—suddenly woke up to find her employer in her bed. To defend herself she passed water, and from that time had suffered from continual incontinence. ERNEST JONES.

HYSTERIA FROM THE POINT OF VIEW OF DISSOCIATED PERSONALITY. (169) MORTON PRINCE, *Boston Med. and Surg. Journ.*, Oct. 4 and 11, 1906, Vol. clv., pp. 372 and 407.

IN this paper, read before the Boston Society for Psychiatry and Neurology, Dr Prince discusses, from a purely clinical standpoint, the relation of hysteria to changes in the personality. Sometimes the physical manifestations of hysteria, the anæsthesias, paralyses, contraction of the visual field, etc., are the most prominent, and sometimes the mental, the moodiness, emotionability, aboulia, etc., but in both cases the essential mechanism—psychological disaggregation—is the same. The disaggregated portion may comprise a separate personality. It is well known that multiple personality is a type of hysteria. But the converse is not well recognised, namely, that hysteria is the disaggregation of the personality, so that the hysteric is a phase of multiple personality.

Prince, in delineating multiple personality as a precise clinical group, has analysed twenty of the best reported cases. He points out that the notion of multiple personality has become strongly but incorrectly associated in our minds with the presence of amnesia, because our conception of the condition has been derived chiefly from those sensational cases in which there had developed an amnesia on the part of one of the personalities for the life of another. This amnesia is, of course, not a necessary phenomenon, and is seen only in pronounced cases. Every hysteric with changes in mood and character is potentially a case of multiple personality.

A typical mode of production of the condition is as follows. An ordinary hysteria develops in the usual way, gradually and without any loss of memory for the previous mental life; the patient slides, so to speak, into a condition exhibiting perhaps hemianæsthesia and contractures with changes in character such as moodiness, irritability, ugliness of disposition, etc. At such a

time no suspicion would arise that we had anything other to deal with than classic hysteria. This condition may persist for many years, or even indefinitely, the patient remembering the whole of his previous mental life. Then, as a result of artificial interference, such as a hypnotising process, or after an emotional shock, the subject may suddenly become completely normal, unification of the personality having occurred. Again no suspicion of anything very unusual is aroused. But now it may be that a sudden and complete relapse to the previous hysteric state takes place; when this happens the hysteric will still remember his early life, but will have forgotten the whole of the experiences acquired while in the normal state. Amnesia is thus added for the first time. Frequently another phenomenon, that of alternation, may occur, so that we thus have a condition of multiple personality established. Prince points out two specially important facts; first, that the early abnormal state may show only mental symptoms and be diagnosed as pure neurasthenia instead of hysteria, owing to the fact that finer dissociation, *i.e.* neurasthenia, frequently, or even usually, complicates hysteria; and secondly, that if this early "neurasthenic" condition has persisted from childhood it may be thought to constitute the normal personality of a congenital psychasthenic, so that the synthetised normal state that supervenes, perhaps in middle life, may be wrongly interpreted as an "abnormal" secondary personality. This incorrect interpretation was until lately applied to several of the celebrated cases of multiple personality, including Félida X. and Marcelline R., and had aroused much speculation and astonishment at the greatly superior nature of this "abnormal" secondary personality. ERNEST JONES.

ON THE QUESTION OF HYSTERIA IN ANIMALS. (Zur Frage der Hysterie bei Tieren.) H. DEXLER, *Neurol. Centralbl.*, Feb. 1, S. 98.

(170)

THIS lengthy and somewhat diffuse article is mainly a critical appreciation of a recent publication of Mainzer's. (See *Review of Neurology*, 1906, p. 513.) The author prefaces this by some theoretic considerations of the mind of animals, particularly of dogs, as contrasted with that of man. He emphasises our ignorance of psycho-pathological processes in animals, which is due to the unhappy separation of neurological and veterinary studies. In general, he is rather opposed to the modern tendency to over-approximate the mental processes of animals and man and maintains that the existence merely of language in the latter sets up an overwhelmingly important barrier. He illustrates his attitude by the following remarks. Epilepsy in animals is almost always a symptomatic secondary occurrence, the commonest cause being an

encephalitis; the existence of this, by the way, can often be determined only microscopically. Gross lesions of the projection system in animals cause psychical symptoms much less frequently than in man, on account of the relative paucity of the association systems in the former. The structure of the brain in animals shows that the differences to be expected between them and men are not only quantitative, *i.e.* on a smaller scale, but also qualitative. He admits, however, that more exact researches in comparative psychology have enabled one to detect more complex psychoses than was at one time thought. Just as word deafness in man has only of late years been distinguished from dementia, so in animals some of the insanities, with "object blindness," etc., can be clearly distinguished from dementia. In dogs it is probable that intense disturbances of taste play at least as important a rôle as deafness in man; and affections of the sense organs are especially difficult to define in animals, just as they are in idiots. Again, an outstanding difference between men and animals is the reduction in the latter of the psychical symptoms compared with the somatic symptoms in such bodily diseases as cancer, severe infections, etc.

Coming now to the question of hysteria in animals the author holds that any affective complaint such as paranoia, typical hysteria, etc., is exceedingly rare if not altogether non-existent in animals. He has seen no such occurrence in the 6000 dogs he has observed for some time. Mainzer's three cases of hysteria he considers of the greatest importance as no former cases had been observed with such detailed analysis by a neurologist of Mainzer's standing. He thinks, however, that the diagnosis of hysteria is a little open to question, principally because of the difficulty in excluding other psychoneuroses, which are certainly commoner. He discusses the three cases in detail, and makes some interesting remarks on the symptom of agoraphobia in general, and of fear of certain localities in particular. He relates several interesting cases in which a definite memory symptom was present so that the animal—dog or horse—exhibited fear on coming to a spot where a previous painful episode had taken place.

ERNEST JONES.

FOOT CLONUS AND BABINSKI'S SIGN IN HYSTERIA. (**Clonus** (171) **du pied et signe de Babinski dans l'hystérie.**) A. VAN GEHUCHTEN, *Le Névraxe*, Jan. 25, 1907, p. 311.

IN a preliminary consideration of the reflexes the author calls attention to a syndrome, the importance of which he has emphasised in previous communications, and to which he thinks other writers have not attached the importance which it deserves. It

is a much exaggerated condition of the deep reflexes upon one side of the body in the absence of any sign of hemiplegia in cases in which a lesion of the opposite hemisphere is probable. He cites the case of a man who, after receiving a blow upon the left side of the head, developed local epilepsy, commencing in the right foot and spreading over the right side till the face was involved. He had no sign of hemiplegia, there was marked atrophy of all the muscles on the right half of the body. The right plantar reflex was of the extensor type. "The tendon reflexes upon the right side were so exaggerated that the lightest percussion with the bent finger upon the tendon of any muscle produced a vigorous contraction of that muscle.

He refers to his classification of the cutaneous reflexes which he published in *Le Névraxe*, Vol. i., 1900. This classification separates the superficial reflexes into two divisions.

1. Abnormal or defensive cutaneous reflexes, perhaps pathological. These require for their production the integrity of the lower reflex arcs (spinal cord) only. They are the reflexes with which physiologists have worked, and they may be called the "skin reflexes of medullary origin" or the "lower skin reflexes."

These are entirely and fundamentally distinct from:—

2. The normal physiological or clinical cutaneous reflexes, which for their production require the integrity of the nervous system.

These he terms the higher cutaneous reflexes.

The author proceeds to argue at length against the somewhat bitter criticisms which Babinski has passed upon his classification and terminology.

Van Gehuchten insists that, as the standard of every normal cutaneous reflex, shall be taken "the minimal motor response to a minimal stimulus." He insists that the normal or higher cutaneous reflexes are obtained only upon stimulation of a definite skin area, and that the response is always in the same muscle. For each of these reflexes there is a pre-existing centre in the spinal cord, with pre-determined afferent and efferent paths.

For the abnormal or lower cutaneous reflexes there are no pre-existing spinal centres. Stimulation of the skin produces ever varying responses, now in one muscular region, now in another. He admits that the most constant and the most easily elicited reaction is Babinski's extensor response, but he points out that the extensor response can usually be obtained by stimulating any part of the leg or thigh. He further emphasises the variability of this lower reflex by citing the fan response of Babinski, the abductor response of Hirschberg and Rose, Oppenheim's reflex, Remak's femoral response and the paradoxical response of Schaffer,

and the flexor response produced on stimulating the skin of the abdomen, and many others.

The detailed description of such phenomena is destitute of any practical value. The important fact which covers the whole ground is the variability of the abnormal or lower cutaneous reflexes as regards form and region, compared with the unvarying character of the normal or higher reflexes.

This variability is a sign that the spinal cord is free from the influence of the higher centres, and that it has returned to the same physiological condition that obtains at a given moment of developmental life, and that is permanently present in the lower vertebrates—the spinal cord becomes a simple reflex mechanism destined for the defence of the individual against excitations which reach the cutaneous surface.

Van Gehuchten then proceeds to the all-important question, Does the appearance of foot clonus, etc., and of the inferior cutaneous reflexes (Babinski's sign, etc.), necessarily point to a lesion of the cortico-spinal fibres? May not these phenomena appear from functional derangements of the cortex, more particularly hysteria? He himself is inclined to a negative answer for the first question and a positive answer for the second.

He cites the case of a young woman eighteen years of age who, after an attack of indigestion with vomiting, suffered with gradually on-coming weakness of the legs, foot clonus, and Babinski's sign on both sides. There was partial hemianalgesia on the right side, the limbs and face escaping. A diagnosis of syringomyelia was made, but a good prognosis was given to the patient with the object of comforting her. Five days later all the signs had disappeared. It is interesting to read that Van Gehuchten had treated this patient three years previously for external strabismus which had recovered after treatment with arsenic.

That such a distinguished neurologist as Van Gehuchten should have based this important opinion on this isolated case is unfortunate, for the antecedent ocular paralysis, in the reviewer's opinion, stamps the subsequent transient spastic paraplegia as the early manifestation of disseminate sclerosis.

Similar cases are by no means rare, and are generally recognised in this country since the publication of many contributions by Buzzard upon this very subject.

The arguments, therefore, brought forward in this paper, that "increase of the deep reflexes and the appearance of the lower cutaneous reflexes with Babinski's sign may be present in exceptional cases of hysteria from psychic interruption of the cortico-spinal fibres," leave the reader very far from being convinced.

JAMES COLLIER.

APPLICATION OF THE DIAZO REACTION TO THE PROGNOSIS
(172) **OF EPILEPSY. (Application de la diazo-réaction urinaire au pronostic de l'état de mal épileptique.)** PAUL MASOIN, *Journ. de Neurol.*, Jan. 20, 1907, p. 21.

THIS article is a repetition of several previous publications on the same subject. In spite of the many adverse criticisms that these have met with, the author hopes that their importance will in time be established. He first discusses the prognostic significance of hyperthermia in epileptic attacks, and agrees with the modern criticisms of Bourneville's former views; the mortality in a series of cases was equal in attacks with and those without this symptom.

He has studied the diazo reaction in detail in 16 cases, and has made the following observations. The reaction was positive in 6 out of 7 fatal attacks, and negative in 3 out of 9 non-fatal attacks; in other words, 6 of the 9 positive cases died, and 1 of the 7 negative cases. From this he concludes that the presence of the reaction is a sinister indication, whereas its absence is of good import. The reaction may appear early, or at any time throughout; the earlier its appearance, the worse is the outlook.

Other observations on the urine during the attacks shewed the frequent occurrence of albumin and acetone, but glycosuria was very rare. ERNEST JONES.

THE RELATION OF ALCOHOLISM TO EPILEPSY. M. WOODS
(173) (of Philadelphia), *Journ. of the Am. Med. Ass.*, Feb. 9, 1907.

THE author discusses the relationship between alcoholism in the parent and the development of epilepsy in the child. From his own experience, he believes that the danger to the prospective child depends not so much upon the parents, one or both, being habitual drunkards, as upon their being intoxicated or under the influence of liquor at the time of conception. Echeverria, after the analysis of about 572 cases of epilepsy, concluded that parental intemperance established a predisposition to epilepsy in 17·30 per cent. of cases, while parental insanity associated with epilepsy antedated but 15·96 per cent. of his cases. Molli finds that from 30 to 40 per cent. of all persons with drunken fathers are epileptics. Martin, after the examination of 150 insane epileptics, found that nearly 60 per cent. had intemperate parents. He also found that in 83 intemperate families with 410 children, 108, or more than one-fourth, were epileptics. Bourneville states that from the examination of 2554 children suffering from idiocy, imbecility, epilepsy, and hysteria, he found that 1053 of them were

the offspring of drunken parents, 983 had drunken fathers, and 80 drunken mothers. Demaux reported 5 out 36 epileptics who were conceived when their fathers were intoxicated. Woods concludes his paper by citing two of his own cases: the first patient, while a temperate man, was the father of two healthy children; later, when he became intemperate, two other children were born, one of which developed epilepsy in his sixth year, while the other had paralytic dementia with epilepsy. In the second instance both the father and mother were moderately intemperate. Of eight children, three were epileptics. C. H. HOLMES.

NEUROSES AND PSYCHO-NEUROSES. (Névroses et psycho-névroses.) (174) F. RAYMOND, *L'Encéphale*, Partie Neurolog., Jan. 1907, p. 1.

PROFESSOR RAYMOND proposes to publish a series of papers on this subject, with the intention, in view of its importance and difficulty, of making a critical exposition of the recent work done, and of defining his attitude towards some of the more disputed problems.

In his preliminary memoir he gives us in a very general way his conceptions of the whole subject. He first insists on the cardinal importance of clinical truths, which are in the last resort the only definite ones, as they stand independent of all explanatory systems. He uses the term functional disease to signify a disease, not without a lesion, but without any *known* lesion. He clearly points out, however, that we are too ready to assume from this that a little more delicate research would demonstrate these lesions. It is very probable that they are not *anatomical* lesions in the gross sense in which we use this word, but *chemical* or *physical;* a magnetised bar of iron is indistinguishable from any other bar in every respect, except for one physical property. This point of view leads to a broader conception of the term organic lesion, a term which raises in our mind ideas of a visible deformity or destruction of certain nervous elements.

Excluding the insanities on the one hand, and certain of the simpler neuroses, such as neuralgia, on the other, he limits himself to the *grandes névroses*, hysteria, epilepsy, hypochondria, psychasthenia, etc. He restricts the term psychoneuroses to the borderland between the neuroses and the insanities (*vésanies*), in contradistinction from the Germans, who include most of the latter in the term. The acceptance of the word indicates the new attitude that recognises the essentially psychic nature of the diseases, and was the forerunner of its natural consequence, psychotherapy. It is only within the past few years that such neuroses as hysteria and the tics have been recognised to be essentially mental diseases.

As to the limits of the group of psychoneuroses, it is interesting to note that, until the appearance of Axenfeld's volume in 1863, such conditions as tabes, hydrophobia, tetanus, etc., were included therein. Many diseases have been excluded since then, and Raymond now suggests that chorea, Parkinson's disease, Grave's disease, and epilepsy be also excluded. In the last instance the group of symptomatic epilepsy has so encroached of late on that of idiopathic epilepsy as to threaten to involve it altogether. He would even exclude from this group of psychoneuroses, hypochondria and neurasthenia, thus leaving only two psychoneuroses, hysteria and psychasthenia. Hypochondria, he considered, should be counted amongst the *vésanies*. The question of neurasthenia is more difficult. Of late years its domain has so extended, including such diverse conditions as hypochondria, rudimentary paranoia, tics, all forms of aboulia, etc., that Tanzi has appropriately named it the giant of neuropathology. "Malheureusement, ce géant n'est pas seulement démesuré, il est informe." Raymond considers that the neurasthenic state, comprising chiefly such symptoms as readiness to fatigue, insomnia, dyspepsia, headache, bad temper, irritability, etc., is only a syndrome that may be induced by numerous conditions, moral shock, infectious disease, organic nervous disease, etc. He would limit it to the neurosis described by Charcot as true neurasthenia, which is essentially an acquired one and curable.

In opposition to this he supports Janet's grouping of what Charcot termed congenital or constitutional neurasthenia, together with the obsessions, phobias, tics, scruples, aboulias, under the name psychasthenia. This is essentially a congenital condition, indications of its presence being manifested in childhood, and, though alleviable, rarely curable.

Fortunately the practical question of treatment does not altogether depend on accurate grouping of the disease, because psychotherapy is fundamentally an individual treatment, varying more with the patient than with the disease. At the basis of all mental treatment comes the question of isolation. Separation from the family and old surroundings is invariably necessary, though the degree of isolation enforced varies from case to case. Raymond, however, agrees with all other modern authorities that the overfeeding and putting to bed employed in the Weir-Mitchell treatment is not the panacea it was formerly thought to be. Hydrotherapy, electrotherapy, and massage are discussed, and their limitations pointed out. Raymond does not think that hypnotism is free from serious inconveniences, so that it should not be employed as a routine measure; in exceptional cases, however, particularly in some forms of hysteria, it is necessary to have recourse to it, as one can in such cases achieve nothing without it.

ERNEST JONES.

ACUTE TRANSITORY PSYCHICAL DISTURBANCES DUE TO
(175) **HEADACHE AND MIGRAINE. (Disturbi psichici acuti transitori su base cefalalgica ed emicranica.)** M. A. BIOGLIO, *Rivist. di Pat. nerv. e ment.*, Jan. 1907, Vol. xii., p. 9.

THE writer has studied these peculiar symptoms described some years ago by Mingazzini, and in addition the slighter disturbances that occur when the pain is less severe; he concludes that in both instances the nucleus of the disturbances is essentially the same. He first describes the following three cases:—

1. Soldier, aged 24. After receiving a sharp remand he was seized with severe headache, which persisted, so that he had to lie down. He then became delirious, with visual hallucinations, and was transferred to an asylum. Cure was complete in two months.

2. Widow, aged 39. She had suffered from chronic headache for many years, with migraine attacks at the menstrual periods. On the occasion of one severe attack she became delirious for eight hours, and had multiple visual hallucinations of a religious nature. This recurred and she was admitted to an asylum. During the intervals she was quite normal.

3. Housewife, aged 37. Since the age of twenty she had suffered from attacks of lancinating and constricting fronto-occipital pains. During the nausea at the height of the attack she frequently experienced visual hallucinations of a simple nature—bright clouds, coloured fires, etc. These became more complex and organised after a psychical attack that followed the birth of her first child.

4. Soldier, aged 21. He was a psychasthenic who had suffered from "petit mal" attacks since childhood. Of late years, especially after the ingestion of alcohol, he suffered from more complicated attacks, in which, after the appearance of violent frontal pain, he lapsed into a state of impulsive agitation—evidently a variety of automatism—and subsequently had profound amnesia for the whole of this period. These attacks were of a hysteric nature.

The author points out the difference between the first group, cases 1 to 3, and the second, case 4. From a study of them, it is clear that acute psychical disturbances may be associated with severe pain in the head, both being due to a common cause, hysteria, epilepsy, etc., or—and this is the main point the author wishes to make—severe pain in the head may be the sole cause of marked psychical symptoms in an otherwise normal person.

ERNEST JONES.

CONTRIBUTION TO THE STUDY OF MENTAL DISORDERS IN
(176) **BASEDOW'S DISEASE. (Contribution à l'étude des troubles mentaux de la maladie de Basedow.)** C. PARHON and S. MARBE (of Bucharest), *L'Encéphale*, Sept.-Oct. 1906.

THE authors report two cases of Basedow's disease with mental disorder. Both patients had a bad heredity; the first was a woman aged 35, the second aged 21. In the first case, the mental symptoms were mental instability, want of attention, irritability, erotic tendencies, hallucinations, ideas of persecution. The goitre had preceded the mental symptoms. In the second case, there were present terrifying hallucinations, distressing dreams, morbid anticipations, and great irascibility. The bilateral resection of the cervical sympathetic improved the physical condition of the second patient, but had no influence on the mental disorder. The authors review the literature on the association of exophthalmic goitre with mania, melancholia, paranoic conditions, conditions of hallucinatory confusion, and hysteria. They refer to two cases published by Boinet, where excessive doses of thyroid extract produced mental disorders such as have been observed in exophthalmic goitre. The authors are of opinion that, in the majority of cases, the mental disorders observed in this disease are due to thyroid intoxication, and are not merely casual associations.

C. MACFIE CAMPBELL.

A CASE OF MUSCULAR DYSTROPHY AND BASEDOW'S DISEASE.
(177) **(Ein Fall von Dystrophia Musculorum progressiva kombiniert mit Morbus Basedow.)** LIEBERS, *Muench. med. Wochenschr.*, Feb. 19, 1907.

THE record is given with full details of the case of a woman aged 23 in whom, since the age of 15, a progressive weakness and atrophy of the face, and of the shoulder-girdle muscles, with pseudo-hypertrophic condition of the deltoid, biceps and triceps, had been coming on. For some months, very distinct symptoms of Basedow's disease (trembling, rapid pulse, sweating, diarrhœa, anxiety, etc.) had come on. The writer regards the two diseases as being accidentally combined, and not causally related to one another; he mentions a similar case recorded by Moebius.

JOHN D. COMRIE.

INSOMNIA FROM DISTURBANCES OF THE PRAEDORMITIO.

(178) **(Die Schlaflosigkeit infolge von Störungen des Vorschlafes.)** KARL LECHNER (of Klansenburg, Hungary), *Klinik f. psych. u. nerv. Krankheiten*, Bd. 11, H. 1, 1907. Carl Marhold, Halle a. S.

THE author in the present paper, which extends over twenty-three pages of this number of the *Klinik*, restricts himself to a preliminary analysis of the normal sleep reflex and a description, illustrated by clinical examples, of the principal forms of disorders of the first phase of sleep (Vorschlaf. Praedormitio). With regard to the analysis of sleep, Professor Lechner finds, besides the "phylogenetically imparted and autogenetically accommodated periodicity," only two incontestable criteria: unconsciousness and recuperation. These are two separate processes, generally but not invariably in direct relation to one another. A long sleep is often accompanied by deficient recuperation and vice versa. Also the depth of the unconsciousness does not always correspond to the resulting restoration, nor even in normal sleep do they run *pari passu*. This is well represented by a chart which has been drawn as expressing the result of numerous experiments, sleep-depth having been calculated by the use of a specially constructed baræsthesiometer and the degree of recuperation by Kraepelin's method (the continued addition of single figures in definite periods of time). Professor Lechner gives two charts which show an abrupt increase in depth of sleep in the first, and an almost equally rapid decrease in the second hour, and later a more gradual wave-like progression to waking consciousness, whereas the restoration curve shows a uniformly undulating elevation; thus illustrating the separable nature of the two processes. The relations of these two events to vasomotor changes are next discussed, and the various forms of disturbances of sleep—described later—indicated, the whole of this part being summed up in the following:—

1. That there are particular disorders of the praedormitio and others of the post-dormitio.

2. That the disorders of sleep depth may be separated from those of the restoration.

3. That according to their heterogeneous (external) or autogenous (internal) origin, sleep disturbances are to be distinguished as exogenous or endogenous.

4. That according to the results of sleep, that is, from the point of view of restoration, sleep disorders may be divided into hyposthenic, hypersthenic, parasthenic, and palinsthenic forms. (By palinsthenic, Professor Lechner means all those in which a slight stimulus is followed by a great positive effect, and moreover, whose resultant does not correspond to the excitation in quality.)

Professor Lechner then proceeds to a minutely detailed description of the quantitative and qualitative disturbances of the fore-sleep, the fundamental disturbances between sleep-depth and restoration being applied to the clinical categories. The whole paper is valuable and in many ways suggestive, but is unfortunately burdened with a novel and most cumbrous terminology.

R. CUNYNGHAM BROWN.

THE KNEE-JERK IN PNEUMONIA. STANLEY BARNES, *Birm. Med.* (179) *Rev.*, April 1906, p. 199.

THE author investigated the knee-jerk in sixty cases of pulmonary disease. In 48 the bacterial cause could be found. Four were empyemata due to various bacilli, in none of which the knee-jerks were affected. The remaining 44 were cases of pulmonary consolidation. In 34 the pneumococcus was found, in 6 the tubercle bacillus, mixed septic organisms and bacteria of decomposition were present in 2, diphtheria bacilli and pneumococci together in one, and diphtheria bacilli alone in one case. The knee-jerks were diminished or abolished in all the cases of diphtheritic or pneumococcal pneumonia, unaffected in septic pneumonia, and unusually active in pulmonary tuberculosis. This condition of the knee-jerk is thus of diagnostic value in pulmonary disease. It has also some prognostic importance, early loss of the knee-jerk indicating profound intoxication. In Barnes's cases loss of the knee-jerk always occurred some time before death if the bacterial cause was the pneumococcus. The date at which the knee-jerks disappeared varied from the first to the seventh day. Return usually occurred two or three days after the crisis. Examination of the third lumbar segment in fatal cases showed that the only changes present were to be found in the large motor cells of the anterior horns which showed all the earlier stages of chromatolysis.

J. D. ROLLESTON.

MARIE'S VIEWS ON APHASIA. F. X. DERCUM, *New York Med.* (180) *Journ.*, Jan. 5, 1907, p. 7.

THE author refers to fourteen cases of aphasia and three cases of pseudo-bulbar paralysis which he has recently examined with the special purpose of testing Marie's affirmations and conclusions. Applying (without criticism) Marie's tests for intellectual impairment, Dr Dercum found in all the aphasic cases above referred to an unquestionable defect, "for all these patients were unable, although in varying degree, to comply with any but the simplest

instructions." Again he remarks, "Defective utterance of such words as were still preserved in the motor aphasics was common to all; though in some it was more pronounced, in others less pronounced." In the only case in which anarthria was entirely absent, there was no impairment of motor speech. He concludes that Marie's contention that aphasia is a unity, an anarthria added to an intellectual defect, is well borne out by the clinical facts. After discussing the question of Brocas convolution, he sums up by remarking that "we are entitled to a legitimate doubt as to the real function of the left third frontal." EDWIN BRAMWELL.

THE "WHITE LINE" OF SUPRARENAL INSUFFICIENCY. (Le phénomène de la ligne blanche, l'hypotension arterielle et l'insuffisance surrénale.) SERGENT et RIBADEAU DUMAS, *Bull. et. mém. de la Soc. méd. des Hôp. de Paris*, July 12, 1906, p. 757. SIREDEY et TINEL. *Ibid.*, Feb. 14, 1907, p. 145. MASSARY et LIAN, *Ibid.*, Feb. 21, 1907, p. 161.

(181)

THE phenomenon designated the "white line" by Sergent is the converse of the *tache cérébrale*, and is due to a spasm of the cutaneous capillaries. It was found by Sergent and Ribadeau Dumas in the various morbid conditions due to suprarenal insufficiency, viz., Addison's disease, certain cases of neurasthenia, intoxications of different kinds, and acute infectious diseases, notably influenza, enteric, scarlatina, and diphtheria. In such conditions there is hypotension and peripheral vaso-dilatation. The mechanical stimulation of the skin produces vaso-constriction and the appearance of the "white line." The phenomenon is best produced by stroking the skin of the abdomen with a finger or other blunt object. Within thirty to sixty seconds a white streak appears, broader than the object which has produced it, and not surrounded by the pink zone characteristic of the white line which appears immediately on stroking the skin of a normal person. The white line persists from three to five minutes, and then disappears. The administration of adrenalin in some cases of diphtheria which presented the white line was followed by the disappearance of the phenomenon.

In a case of tuberculous meningitis recorded by Siredey and Tinel in a man of 47 the appearance of the white line in place of the *tache cérébrale* suggested a lesion of the suprarenals. The blood-pressure was low, but there was no abnormal pigmentation of the skin or mucous membranes. The forecast proved correct, for the autopsy revealed not only tuberculous meningitis with pulmonary and peritoneal tuberculosis, but also large tubercles in the suprarenals. The liver and kidneys were not affected.

Massary and Lian have recently reported two cases of insolation, in one of which they observed the white line, while in the other the *tache cérébrale* appeared. The two phenomena were regarded as due to the same cause, both being the result of meningeal irritation. The white line, according to Massary and Lian, is simply the result of a nervous disturbance, which may be slight and purely functional (physiological white line), but which in disease is due to a primary or secondary alteration of the nerve centres. This alteration is primary when the nervous system alone is involved, and secondary when the nervous system is affected by various morbid influences, among which must be included perturbation of the functions of the suprarenals.

J. D. ROLLESTON.

PSYCHIATRY.

(182) **THE CLINICAL ASSESSMENT OF MEMORY IN NERVOUS AND PSYCHICAL DISEASES. (Ueber Art und Wert klinischer Gedächtnismessungen bei nervösen und psychischen Erkrankungen.)** PAUL RANSCHBURG (Budapest), *Klinik f. psych. u. nerv. Krankheiten*, Bd. 11, H. 1, 1907. Carl Marhold, Halle a. S.

SINCE the publication of his *Studien ueber die Merkfähigkeit* in the *Monatsschr. f. Psych. u. Neurol.* of 1901, reference has been frequently made by other workers who have used it to Dr Ranschburg's "word-pairing" method. This consists essentially of presenting to the subject, either orally or exposed upon a screen, paired words, and then, after a definite interval, presenting the first word of each pair and testing the power of recall of the second. As elaborated by Dr Ranschburg, the method is of great importance both from the point of view of pedagogical inquiry and of psychiatry. The author limits the present paper to a general description of his method and its application to nervous and mental cases. Under definite and uniform conditions as to method, time, and length of exposure, character of verbal material, and duration of interval between the exposure and examination of the subjects, the memorial product can be expressed in terms approximating to mathematical accuracy. The whole paper, which is not completed in this number of the *Klinik*, is so compact with information that it can hardly be described in less space than itself.

R. CUNYNGHAM BROWN.

ATTACKS IN DEMENTIA PRÆCOX. (Les ictus dans la démence
(183) **précoce.)** PASCAL, *L'Encéphale*, Sept.-Oct. 1906.

THE author discusses the question of the occurrence of epileptiform and apoplectiform attacks in dementia præcox, reviewing the opinions of previous authors. The occurrence of epileptiform convulsions raises the question of the relation of such cases of dementia præcox to hysteria on the one hand, and to epilepsy on the other. Where the characteristic deterioration of dementia præcox occurs in well-marked cases of hysteria or of epilepsy, with attacks dating far back, the author concludes that there is merely an association of the neurosis with dementia præcox. But convulsive attacks may occur in cases of dementia præcox which have shown no other evidence of hysteria or epilepsy, and where the attacks must be attributed to the basis of the psychosis itself. The relation of the attacks to the psychosis is variable. The attacks may precede the psychosis by a long period; they may first occur after the psychosis has been well established, or they may immediately precede the development of a rapid dementia. The attacks in dementia præcox are usually convulsive in nature; apoplectiform attacks are rare. The difference between the attitudes of various authors is shown by the fact that the author diagnoses as dementia præcox two cases with convulsive attacks, which Janet has reported as cases of hysteria and psychasthenia. As to the pathogenesis of these attacks, we know practically nothing. C. MACFIE CAMPBELL.

ACUTE HALLUCINATORY CONFUSION AND HEPATIC
(184) **INSUFFICIENCY. (Confusion hallucinatoire aigue et insuffisance hépatique.)** G. DENY and M. RENAUD (of La Salpêtrière, Paris), *L'Encéphale*, March-April 1906.

THE patient was a woman of 38, with no psychopathic disposition, neither syphilitic nor alcoholic, who after several years of gastric disorder, rather quickly developed a condition of hallucinatory confusion with vague ideas of persecution, fears of poisoning, and general apprehensiveness. The clinical examination of the viscera was negative, the bowels were irregular, the tongue dry and coated; patient quickly became feebler; diarrhœa developed, and there was evidence of intestinal hæmorrhage. The autopsy disclosed an atrophic condition of the small intestine, with some small hæmorrhagic areas; the kidneys showed diffuse parenchymatous alterations; the liver showed very marked general fatty degeneration; the central nervous system showed well-marked cellular changes; there was no inflammatory element present. According

to the author, the following is the interpretation of the case; the patient had a slight gastro-intestinal affection; the liver was unable to exercise its normal rôle, and the mental disorder followed upon this hepatic insufficiency. The renal insufficiency accentuated the auto-intoxication. Clinically, the mental disorder was almost the only expression of the hepatic insufficiency.

C. MACFIE CAMPBELL.

A CONTRIBUTION TO THE PSYCHOLOGY OF LEGAL EVIDENCE.

(185) **(Beitrag zur Psychologie der Aussage. Auf Grund eines gerichtlichen Gutachtens.)** J. HAMPE (of Brunswick), *Klinik f. psych. u. nerv. Krankheiten*, Bd. 11, H. 1, 1907. Carl Marhold, Halle a. S.

IN this paper Dr Hampe discusses at considerable length one of those cases, frequently before the bar of justice, where the responsibility of the accused is difficult to determine, where facts and arguments which appear cogent to the psychiatrist are dismissed as unimportant or irrelevant by the jurist, cases for which the law does not as yet make adequate provision and in which a just sentence—or what seems so to the mental expert—can only be obtained by an elastic, and to the legal mind unwarrantable, interpretation of the law.

Briefly the main facts are as follows:—S. the accused, who had already served one or two terms of imprisonment, was tried at the Assizes for perjury, having given false witness during the trial of his son for the assault of a man with whom both father and son were at enmity. Doubts as to his sanity were entertained during his remand whilst awaiting trial, but no prison officials were called to give witness, and although similar doubts had been expressed on a former sentence in 1884, evidence of this was ruled out as inadmissible through the death of the persons concerned. At the trial the following facts were elicited: hereditary influences (mother demented, second son weak-minded); S. himself a sober, industrious, and on the whole well-behaved man, but from childhood somewhat simple and below the average mentally; thirteen years before this trial, head-trauma with loss of consciousness, followed by various nervous symptoms, headache, depression, insomnia, impaired memory, at times thoughts of suicide, and periodic fluctuations of emotional tone. Alcoholic intolerance is also asserted by Dr Hampe, but this apparently does not rest on an experimental basis. On bodily examination, apparently normal. Examination as to the psychical state showed marked intellectual incapacity, very deficient power of attention, incomprehension of abstract terms, including the meaning of "perjury"; and examina-

tion by Ranschburg's word-pairing method showed a paramnesia from frequent confusion of past with recent impressions. On the grounds of the above results Dr Hampe has no hesitation in including S. within the scope of § 51 of the Strafgesetzbuch. In the absence, however, of specific delusions or hallucinations, the necessity for whose presence seems to obsess the legal mind, S. was sentenced to five years' penal servitude.

R. CUNYNGHAM BROWN.

(186) **THE REPORT OF THE 1906 CONVENTION FOR LEGAL PSYCHOLOGY AND PSYCHIATRY FOR THE DUCHY OF HESSE. (Vereinigung für gerichtliche Psychologie und Psychiatrie im Grossherzogtum Hessen.)** *Juristisch-psychiatrische Grenzfragen*, Bd. 5, H. 6. Carl Marhold, Halle a. S., 1907, pp. 66, price M. 1.20.

THIS report of the proceedings at the above congress, held under the presidency of Attorney-General Dr Preetorius of Darmstadt at Butzbach last July, is composed of two parts: the first dealing with a discussion upon the organisation of courses of lectures upon penology for jurists; and the second with the function of medical, and particularly of alienist, experts in the witness-box. The discussion on the organisation of lectures on penology (Gefängnislehrkursen) was opened by Professor Mittermaier of Giessen, who strongly maintained that such courses should not only include lectures on penological principles, on psychology, anthropology, sociology, and statistics, as theoretical subjects from the special point of view of criminology, but also a practical course. The first, the theoretical, appertained to the University, but the practical course could only be carried out in the prison, wherein not only the conditions of actual punishment could be studied, but the criminal himself. The following speakers were in substantial agreement with Professor Mittermaier, and the resolution was passed that, subject to the consent of the Minister of Justice (Lord Chancellor), courses (in penology and criminology) should be inaugurated in Hesse for jurists and medical men in the service of the State; the practical course to consist of ten to fourteen days a year, and to be conducted at the central prison of Butzbach and Marienschloss, preceded by theoretical instruction.

Such practical instruction is already given at Baden (since 1886), in Prussia (since 1895), in Bavaria (since 1900), and in Hamburg (since 1904), and there seems to be no reason why it should be limited to these places, or indeed to Germany, for there can be no doubt, as Professor Mittermaier said, that an intimate knowledge of the factors of crime can be gained more easily and completely

in the prison than in the law courts; that an acquaintance with the conditions and effects of punishment—surely indispensable to the judge—can be acquired only in the prison; and that the prison affords opportunities for such study of the working of the criminal mind as would be invaluable to those who have to settle the question of the responsibility or irresponsibility of persons accused of crime. This leads at once to the questions: Who have to settle the responsibility or the irresponsibility of the accused? and, What is the place and function of the medical witness, and in particular of the alienist expert? As to the former question, the law as it stands, in Germany as in England, admits of no doubt. The settlement of the responsibility or irresponsibility of an accused person rests with the judge and jury and never with the medical expert. This was of course the view of Professor Mittermaier, who introduced the discussion, *Die Tätigkeit des Medizinischen, im besondern des psychiatrischen Sachverständigen vor Gericht*, and this was acquiesced in by all the subsequent speakers, including Professor Sommer, who restricted his remarks to a criticism of existing practices as to the preparation and framing of evidence for judgment. He stated, however, that, since the time when the laws were formulated, concepts as to what constituted irresponsibility have largely altered, and that the law should be interpreted in the light of modern information, *i.e.* should regard the results of minute psychological inquiry into the total personality of the individual and, by implication, the extension of the protective clauses (§ 51, Str. G.-B.)[1] to persons not strictly and legally within their scope, or who were not considered so when the laws were framed. It is evident, however, that if this question of responsibility is one solely for the jurist, as it undoubtedly is at present in legal courts, and if the expert evidence is to be rigidly confined to facts and not to include expert decisions, then the jurist must be in a position to appreciate the bearing of every one of these facts; that is, he must be instructed in psychiatry. This is the position taken up by Professor Sommer, who prophesies that in the near future "a certain psychiatrical knowledge will be obligatory upon the jurists," in support of which he refers to a recent ministerial order. R. CUNYNGHAM BROWN.

[1] § 51 of the *Deutsches Strafgesetzbuch* runs as follows: "Eine strafbare Handlung ist nicht vorhanden, wenn der Täter zur Zeit der Begehung der Handlung sich in einem Zustande von Bewusstlosigkeit oder krankhafter Störung der Geistestätigkeit befand, durch welchen seine freie Willensbestimmung ausgeschlossen war."

THE DEVELOPMENT OF THE FAMILY-CARE OF THE INSANE
(187) **IN GERMANY SINCE 1902. (Weiterentwicklung der familiären Verpflegung der Kranksinnigen in Deutschland seit 1902.)** KONRAD ALT. Carl Marhold, Halle a. S., 1907, p. 16, price 75 Pf.

IN this short paper contributed to the International Congress of Alienists held at Milan in September last, Professor Konrad Alt, the well-known director of the great institution for the insane at Uchtspringe, Saxony, gives a comprehensive account of the growth of the family-care system in Germany in recent years. This growth, though rapid, will not appear surprising to those who are conversant with the admirable family-colony system which mostly obtains in Germany. The figures which Professor Alt gives speak for themselves. Whereas in 1882, in the whole of Germany, there were only two family-colonies, with about 50 boarded-out insane, in 1902 the colonies had increased to 32, with about 1200 patients. Since that time the proved value of this mode of disposal of the insane, both as a means of treatment and as a pecuniary gain, has won for it the confidence of both alienists and administrative authorities, with the result that, since 1902, the family-care system has been adopted as an auxiliary to asylum treatment at 19 new centres, the insane so treated have risen to 2400 in number, and fresh developments are everywhere under consideration. Professor Alt's survey is accompanied by a map of Germany showing the distribution of the family-colonies in Germany, and a chart showing graphically the growth of the boarded-out insane in Saxony in the decennium 1896-1906, showing a slow increase in number from 2 in January 1896 to 51 in 1900, and a rapid rise from that date to 475 on January 1, 1906. Attention need be drawn to only one other part of this paper. Medical superintendents in this country have occasionally expressed the opinion that the withdrawal of the well-behaved and able-bodied patients from the asylum to the outer colony must seriously affect the working capacity of the asylum. The same fear, Professor Alt says, has been expressed by some writers in his country, but his ten years' experience has convinced him that the system does not necessarily entail any such result. The patients sent to family-care need not be composed only of good workers. Many who are idle in the asylum work cheerfully under family-care, and the withdrawal of numbers of good workers from the asylum at Uchtspringe has never permanently lowered the asylum output of work, the vacancies always being filled up by the remainder, even though previously unemployed. The point is of purely fiscal importance, for obviously the asylum exists for the insane

and not the insane for the asylum, and experience has abundantly shown that the asylum *plus* the colony is much more economical than the asylum alone. R. CUNYNGHAM BROWN.

THE CYTOLOGICAL EXAMINATION OF THE CEREBRO-SPINAL
(188) **FLUID IN CASES OF MENTAL DISEASE. (L' esame citologico del liquido cefalo-rachidiano nelle malattie mentali.)**
PEGNA, *Ann. di Nevrolog.*, f. 4, 1906, p. 241.

THE writer, in an article extending over 50 pages, quotes many authors to show that a lymphocytosis is a diagnostic feature of general paralysis, and he himself studies 65 cases of insanity, including 12 of this disease, which confirm this view. He further brings out a fact of the greatest importance in proving that the cerebro-spinal fluid of general paralytics is toxic to guinea-pigs. He divides his cases into three groups and subjects them to a series of examinations which comprises the specific gravity, cryoscopic index, percentage of lymphocytes and of serum globulin, presence or absence of serum albumin, negative bacteriological findings and toxicity.

Groups 1 and 2 contain 46 cases of all varieties of insanity, including epileptics. From examination of these he concludes that in no variety of insanity is there lymphocytosis except where there is a history of syphilis, and then it is only "discrete," *i.e.* only few cells present. The cerebro-spinal fluid is toxic to guinea-pigs only in the case of epileptics while suffering from fits, and the toxity is proportionate to the severity and duration of the convulsions.

Group 3 consists of 7 cases of senile dementia and 12 of general paralysis, and in these he found a trace of serum albumin in the former when arterio-sclerosis was present. While in general paralysis lymphocytosis always exists at some stage of the disease, serum albumin is present in varying amount according to the number of lymphocytes. The percentage of serum globulin is always high, and the specific gravity is increased (1009-1010). The cryoscopic index varies much in general paralysis, but is as a rule much lower than in other forms of mental disease.

The effect of general paralytic cerebro-spinal fluid on guinea-pigs was very pronounced, varying in intensity with the proximity of congestive seizures. The inoculated animal died always within ten hours, and in one case in half an hour.

The author emphasises his conclusions in an interesting summary. He states that a lymphocytosis of fifty to ninety in the cerebro-spinal fluid means an organic affection of the nervous system involving the meninges, and so serves to distinguish it from functional disorder, with a precision which cannot be done

by any other known clinical method. A negative result doesn't exclude general paralysis, but in the absence of lymphocytosis one would make a diagnosis of the disease with the greatest circumspection, since it is usually one of the first signs of the malady. Cases of insanity in which there is a history of syphilis may show a lymphocytosis, but only in a very minor degree.

The cryoscopic index in cases of general paralysis is usually low. The high percentage of serum globulin and the presence of serum albumin are valuable diagnostic indications.

Toxicity of the cerebro-spinal fluid to guinea-pigs, inoculated with a quantity (five per cent. of body weight) of the material, is diagnostic of general paralysis, except only in cases of epilepsy during an accession of fits. DOUGLAS M'RAE.

TREATMENT BY EMPLOYMENT FOR NERVOUS CASES.

(189) (**Beschaeftigungstherapie für Nervenkranke.**) LAEHR, *Wiener kl. Wochenschr.*, Dec. 27, 1906.

IN this paper the writer discusses the practical value of the treatment of cases of nervous disease by means of work, and gives the results of six years' experience as director of a sanitorium near Berlin.

He discusses what is meant by the term "work," and decides that the criterion of suitable work is that the person shall feel happy in doing it. After such work the most lasting and intense change of mood takes place; and, generally speaking, the proper work is of a physical nature. This form of treatment, he states, adapts itself to any form of neuropsychosis, whether manifesting itself as neurasthenia, hysteria, or hypochondriasis, and is specially suited for traumatic cases. Two primary conditions must be present, however: that the patient must feel ill and wish for treatment, and that he must have no insuperable disinclination to work. The quickest and best result is obtained, he says, from those patients who realise most fully the importance of the work for their bodily and mental improvement. Contra-indications for the treatment are an acute condition of exhaustion, toxic conditions, weakening diseases, some injuries, and great overstrain.

As to the nature of the work, he believes that manual work of a purposive character in the open air is most suitable for all classes. Above all, he recommends carpentry and gardening; for weaker persons bookbinding, cardboard work, and wood-carving; sometimes drawing, painting, or photography; and for women various kinds of housework. With the patients there must be a skilful, sympathetic, and tactful supervisor, and the physician must carefully watch the effect of the work upon the patient.

JOHN D. COMRIE.

THE ACTION OF SCOPOLAMINA IN CHOREA. (De l'action de
(190) **la Scopolamine sur la Chorée de Sydenham.)** BABINSKI, *Soc. de Neur.*, Jan. 10, 1907, *Rev. Neurol.*, p. 86.

BABINSKI has treated four chorea patients with hydrobromide of scopolamine given subcutaneously in doses from $\frac{1}{4}$ to $\frac{1}{2}$ mg. a day. The results were good in all cases; in one it was very striking as the disease was very severe and prevented sleep.

ERNEST JONES.

RESULTS FROM THE SURGICAL TREATMENT OF BRACHIAL
(191) **BIRTH PALSY.** ALFRED S. TAYLOR, M.D., *Journ. of the Am. Med. Ass.*, Jan. 12, 1907.

BRACHIAL birth palsy of the Erb type is due to overstretching of the nerve roots, resulting in rupture of the sheath and nerve fibres, hæmorrhage between the torn fibres, and resulting cicatrisation which prevents transmission of nerve impulses. Nerve conduction can be re-established only by excision of the cicatricial area or areas, and end-to-end suture of the nerves. The nerves involved having been ascertained before operation, the brachial plexus is exposed at the lower part of the posterior triangle of the neck; the adherent fascia is dissected off, the indurated portions of the nerves removed, and the healthy ends joined by suture, or, if the gap is too great for this, by means of a catgut bridge. Though the suprascapular nerve is small, it must receive special attention, as it supplies the external rotator muscles of the shoulder, whose paralysis causes the marked internal rotation of this paralysis, as well as the tendency to posterior dislocation of the shoulder.

Early operation—between six and twelve months if possible—is indicated. Delay results in impaired development of the extremity, contractures of muscles and ligaments, and atypical shape of the joint-ends of the bones, all of which diminish the chance of operative success. Massage, electricity, and passive movements must be employed to prevent these deformities both before and after operation. Generally additional muscles are at first paralysed by the operation, as some normal nerve fibres are necessarily divided; but this operative paralysis has often completely disappeared within six or eight months. Improvement in nutrition and development of the limb usually begins in four months, and return of power in the originally paralysed muscles appears in from six to ten months. The author has operated upon nine cases. Two died from causes not inherent in the operation itself. One case, in which nerve-bridging had to be employed, showed trophic disturbances of the fingers, which comparatively soon disappeared. All the cases which recovered show considerable and progressive improvement both in development and function.

W. J. STUART.

Reviews

A TEXT-BOOK OF PSYCHIATRY. LEONARDO BIANCHI, Professor of Clinical Psychiatry and Neuropathology in the Royal University of Naples. Translated from the Italian by JAMES H. MACDONALD, M.B. Pp. 877. London: Baillière, Tindall & Cox. 1906.

THIS is a lengthy volume, of which, roughly, nearly one-half is devoted to a description of cerebral anatomy—macroscopical and microscopical — and physio-pathology. The remaining larger portion deals with clinical psychiatry. As a matter of experience we think that this combination of the two subjects in one book, however important their bearing the one upon the other, is a mistake. The modern student, driven by the force of a stupendous pressure to systematise knowledge and to acquire facts, has neither the time nor the inclination to sit down to the study of a compendium embracing at once everything that can possibly be known about one system of the body; he likes his information in departments; if clinical, then he applies to a clinical work; if physiological, he prefers the laboratory or a work by a physiologist. It is the same with the busy physician.

The work of Professor Bianchi upon the sciences cognate to and introductory to psychiatry are so well known, and his erudition and research are so well established, that the first portion of this work need not detain us at the present time. It contains a treasury of useful information, luminously presented and inclusive of most of the facts which a student of neuro-physiology and neuro-pathology requires in his reading. The direct bearing of these facts upon the teaching of clinical psychiatry has not as yet been by any means established. This is not stated by way of reproach to our author, but as an unfortunate though irrefutable fact.

The part of the book devoted to the description of mental diseases is very clearly written, and contains much valuable information which, moreover, bears the unmistakable stamp of originality and of long and patient observation. It is unfortunate that the usefulness of the book as a work of reference is somewhat curtailed by the want of any definite line of classification, and occasionally by the use of a terminology which is alien to any recognised nomenclature in the literature of the subject. In his discussion upon classification, Professor Bianchi remarks that "The two criteria in the formation of a scientific classification which have been so happily employed in general medicine would, if adopted in psychiatry, give a very inferior and confusing result. I allude to the ætiological and anatomo-pathological criteria." It is strange that in this connection he neglects to mention the symptomatological criterion which is so happily combined in

general medicine with the other two. Why should it not also be combined in psychiatric medicine? In any case the author does not incline to adopt any classification, but contents himself by dividing all mental affections into three groups as follows: (1) Affections represented essentially by an evolutionary psycho-cerebral defect; (2) A group comprising all the mental affections of infective, autotoxic, and toxic origin developing in individuals regularly evolved; and (3) All mental infections with an organic substratum, localised or diffuse, in the central organ of mind. The scheme is ingenious and plausible, but a little consideration will show that the result cannot fail to be unsatisfactory owing to the incessant overlapping of the various members of the different groups. A cursory glance at the classification is apt to be bewildering in the extreme, for it reveals the close proximity of clinical entities which we have always been taught to regard as essentially distinct and unrelated.

With regard to terminology, many points might be referred to, but one will be sufficient. Professor Bianchi sets up a new clinical entity which he designates "Sensory Insanity." He tells us that he makes use of this name because in his experience the first symptoms of the disease invariably commence with sensory disorders (hallucinations or illusions). The initiated reader will perceive before he has read very far into this disease that he is dealing with Kraepelin's Dementia Præcox, every division of which is described and included. It is satisfactory to find two such distinguished physicians confirm one another's clinical experiences. On the other hand, it is unfortunate for himself that Bianchi's claim for priority is so hopelessly in arrear, although we confess that we should find it difficult to adopt his theory of sensory origin for the symptoms of this great group.

Generally speaking, Professor Bianchi's book is, as we should expect from the pen of so eminent a man, a perfect mine of new and instructive information, and if it is not arranged to suit the tastes of the British reader, we are willing to concede that that may be quite as much the fault of the Islander as of the Italian.

In any case it is not the fault of the translator, who has devoted evidently a great amount of time and thought to presenting to the English-speaking reader, in commendably lucid language, the first great Italian work on mental diseases that has been translated into English.

DIE POLIOMYELITIS ANTERIOR ACUTA. GEORGE FLATAU. Benno Konegen, Leipzig, 1906, price M. 1.

THIS pamphlet of 26 short pages, which has been sent us for review, is one of a series dealing with the more important diseases of the nervous system, and written expressly for the general practitioner. EDWIN BRAMWELL.

Proceedings of Societies

AT a meeting of the New York Psychiatrical Society, held on January 2, 1907—the President, Dr Adolf Meyer, in the Chair—a paper on "The Symptomatic-Prognostic Complex of Manic-depressive Insanity" was read by Dr George H. Kirby, of the Pathological Institute of the State of New York. Dr Kirby said: The field of psychiatry is too complex for us to expect absolute or clear-cut distinctions either in disease types or nosological groups. Kraepelin did a great service by showing that most of the previously made distinctions of types could be replaced by far more valuable nosological groups if one considered the whole *course* and *outcome* of mental disorders. From this point of view the great bulk of the acute psychoses can be brought under two divisions, viz. those that pass into deterioration and those that end in recovery. It was found that in cases which terminated in either of the above mentioned ways, one was able to identify certain characteristic symptoms *present from the beginning*, and which thus acquired a distinct prognostic value. Two large symptomatic-prognostic groups were thus created—Dementia Præcox and Manic-depressive Insanity. At present it seems best to consider the manic-depressive complex merely as a reaction type and not a disease entity as Kraepelin proposes. It is rather a type of response which may be elicited in various ways. In some cases the constitutional disposition may be most important, but in others the exogenous causes are of great importance and give valuable hints as to prophylaxis and probability of recurrence. This peculiar kind of reaction in its pure form has the characteristics of a benign disorder and thus a favourable prognosis is implied. Various additional elements may enter to confuse the picture, and the symptomatic and prognostic features may seem to diverge at various times. There are many equivalents difficult to recognise. In a few cases the principle seems to fail through transitions to other forms.

A series of cases was reported which had offered difficulties for various reasons. Some patients were also presented. A recurrent depression with retardation and prominent auditory hallucinations. Another case of depression with sensory-somatic complex and only slight signs of difficulty in thinking. A third case illustrating the mixed forms of manic-depressive insanity, the so-called "manic-stupor," showing inhibition and mutism with an exhilarated mood.

DISCUSSION.

Dr Charles L. Dana was inclined to think "prognostic principle" a rather unfortunate term, inasmuch as this was not all that was used in attempting to establish types. The consideration of the

whole life history was the great principle to be used in making classifications, and he did not think there could be any objection found to basing groups of insanity upon this method. It was not new, but the rational and accepted one in other forms of disease. The case presented by Dr Kirby in which there was melancholia without retardation of thought was interesting, but was a type not infrequently seen outside of asylums at least, and often in connection with intense suicidal feelings. This suicidal impulse was not always due to somatic feeling of inadequacy, but was sometimes also a well reasoned out point of view. A woman of intelligence, for example, realising that she had had melancholia now for three times, that she would have it again, that she would be a burden to her family, that she would never be right permanently, schemes to kill herself, obeying in a way a logical conclusion as well as a morbid impulse. Such patients did not have always retardation of thought, but they often could not do their work easily or effectively. He did not believe the first patient presented by Dr Kirby would be able to do her work. Such patients could not play games of skill as formerly, becoming tired quickly. Dr Kirby had neglected to touch upon chronic melancholia, melancholia of involution, a group which Dr Dana believed to be represented in early life. He had records of cases of melancholia at the climacteric who gave a history of depression earlier very much like that of later life, so that there was a melancholia of early life which did not belong to manic-depressive insanity.

Dr L. Pierce Clark thought the symptom-complex of manic-depressive insanity would be easier of analysis if the idea were borne in mind that the complex was likely to partake of the nature of the physiological epochs at which it occurred. The suicidal impulse to which Dr Dana referred was well known to most alienists. Even in convalescence this tendency was most marked.

Dr August Hoch said it was usually claimed that it was impossible to formulate any laws of prognosis about the different forms of manic-depressive insanity. This was not quite correct. Dr Kirby had shown that a careful reasoning might do much in formulating a special prognosis. Dr Hoch wished to mention a few more general points of view from which a prognosis might be made. The first referred to the depressions. The symptoms of the entire depressive complex were retardation, sadness, and the anxiety-unreality complex. In the typical manic-depressive states there were only sadness and retardation. In such cases the outlook was good. In the typical involution melancholias there was, from the beginning, uneasiness, anxiety, to which were often added later in the course symptoms of the anxiety-unreality complex. In such cases the outlook was bad; such patients got into a state of deterioration, characterised by a narrowing of the

mental horizon. But the anxiety-unreality complex might also be reached by way of the manic-depressive states. This happened not infrequently in the manic-depressive depressions, which occur at the involution period, sometimes in those occurring earlier in life. These cases might, therefore, begin with a typical retardation or feeling of inadequacy, and then develop an anxiety-unreality complex. In such cases the prognosis was not so bad as in the typical involution melancholias, but decidedly less good than in the simple manic-depressive depressions.

The second point of view referred to the prognosis of the manic states. All alienists knew that those cases which present the .best prognosis are cases of clean-cut manias of considerable intensity, manias in which the exhilaration, the general excitement, and the flight of ideas are all of about the same degree. The hypomanic states were much less favourable prognostically. Dr Hoch had always found that cases in which the disorder of behaviour was out of proportion compared with the flight of ideas and the intellectual disorder in general, were prognostically also more unfavourable. Such cases were apt to have long attacks or many attacks. The prognosis in cases in which a delusional element was strikingly out of proportion to the flight of ideas was also more doubtful than in the simple classical mania. In other words, the cases of mania in which there were strong discrepancies in the intensity of the different symptoms were those which presented a less favourable outlook than those in which there existed a certain harmony in the intensity.

C. MACKIE CAMPBELL.

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AN INTERNATIONAL CONGRESS OF PSYCHIATRY, NEUROLOGY, PSYCHOLOGY, AND THE NURSING OF LUNATICS

will be held at Amsterdam on the 2nd to 7th September 1907, under the patronage of Her Most Gracious Majesty Queen Wilhelmina and of His Royal Highness Prince Hendrik of the Netherlands. The Congress will be divided into two parts: General Sessions and Meetings of Sections. In the former, chosen subjects will be dealt with by competent men who have been invited for that purpose; in the latter, questions will be discussed and criticism will be invited. Anyone taking an interest in Psychiatry, Neurology, Psychology, and the Nursing of Lunatics may become a member of the Congress. Those wishing to attend the Congress are requested to communicate with the General Secretary, Prinsengracht 717, Amsterdam.

Review
of
Neurology and Psychiatry

Original Articles

A CLINICAL AND EXPERIMENTAL INVESTIGATION INTO THE LYMPHOGENOUS ORIGIN OF TOXIC INFECTION OF THE CENTRAL NERVOUS SYSTEM.[1]

By Dr ORR,
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and
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In a previous paper (1) we described the lesions in the posterior columns of the spinal cord in general paralysis, and pointed out their similarity with those in early tabes dorsalis. Our conclusion with regard to the starting-point of the degeneration was that it commenced in the intramedullary portion of the sensory fibres immediately the neurilemma sheath was lost. It is important to clearly differentiate this point from that at which the posterior roots undergo a constriction on passing into the cord, as they do not coincide; in fact in the region most usually concerned in tabes (lumbo-sacral) the neurilemma leaves the fibres at a point situated outside the constriction (2). Nevertheless we would speak of the nerves as intramedullary as soon as they lose Schwann's sheath.

During these observations on the sensory fibres, in which it was apparent that the absence of neurilemma played an important part in the localisation of the onset of the lesion, we

[1] Towards this research the British Medical Association has assisted by a grant of money.

naturally inquired into the condition of the intramedullary portion of the motor nerves, but found the changes in these as a rule difficult to trace. We therefore extended our investigations to the cranial nerves, and found that the lesions began in both sensory and motor roots at a point which corresponds to that already indicated in the spinal nerves; and the changes found therein form the first part of this paper.

While our attention was still confined to the posterior columns of the cord we had indications of the advisability of inquiring into what was already known of their lymphatic system, and we obtained evidence of a continuous flow of lymph upwards along the nerves, and that the main current lay at the periphery of the nerve bundles immediately under the fibrous sheath. References to the authors holding this view have been given in our previous publications, so that here a short recapitulation will suffice. It is well known that tetanus and rabies spread to the cord by the nerves; and in this connection we might mention the experiments of Marie and Morax (3) who, after cutting the nerve to the fore limb of an animal and later injecting a lethal dose of the tetanus toxin into the paw, found that no convulsions followed. Homén and Laitinen (4), after injection of streptococci into the sciatic nerve, traced the organisms upwards into the meninges of the cord; while Pirrone (5), experimenting with the pneumococcus, found changes in the cord, but limited to the side corresponding to the nerve injected. In addition to organisms, chemical and inert substances have been used with like results, *e.g.* Guillain (6) injected ferric chloride into the sciatic nerve, subsequently introducing potassium ferrocyanide into the general circulation, and found Prussian blue in the posterior roots. Sicard and Bauer (7), using China ink, observed, after injection into the nerve, that the granules ascended along it towards the cord. It was presumable therefore that, if these views were correct, we ought to find in the cord of cases in which some septic focus existed, lesions of the posterior columns caused by toxins ascending in the lymph stream. On examining cases of brachial neuritis (infective), bed sores, suppurating knee-joint, septic psoas abscess, we found, in the cord of all, the lesions expected. These also commenced at the vulnerable point above indicated, and resembled in their distribution early tabetic degenerations (8).

Bearing these facts in mind, the question of a possible lymphogenous infection of the central nervous system seemed worthy of further investigation, owing to the apparent facility with which toxins gained access by the nerves; and it was with a view to testing this that our experiments were conducted. Besides, apart from the question of etiology, the introduction of toxins by the nerve lymph paths promised, if successful, to offer a convenient means of inducing cord and cranial nerve lesions, and to afford one the opportunity of studying them in their early phases. We have already published a series of clinical cases (1 and 8) showing the lesions found in the spinal cord in general paralysis and in other toxic conditions, and before giving the results of our experiments we submit the following cases illustrative of lesions of the cranial nerves.

Lesions of Cranial Nerves in Toxic Conditions.

Clinical Cases.

Case 1.—An ataxic general paralytic. The first symptoms were noticed about three years before his death.

Examination of the pons and medulla showed the following lesions of the *cranial nerves*:—

Third Nerve.—Both third nerves were affected to a slight degree. By the Marchi method the degeneration was exhibited in the form of small, round, black globules, which could be followed along the nerve fibres from the periphery almost to the nucleus of origin.

Fourth Nerve.—The left fourth nerve was affected much more than the right. In the latter, globules were present, as in the third nerve, but were larger. In the left nerve the degeneration was in the form of larger and more irregular masses, which were arranged in series along the nerve fibres. They could be traced from the point where the fibres lost their neurilemma sheath—and the lesion was most intense in this region—to the nucleus of origin. There was no degeneration in the fibres outside the pons.

Fifth Nerve.—The left fifth nerve showed more degeneration than any other cranial nerve, and exhibited many interesting changes; the right was also affected, but to a less degree. Immediately beyond the point where the fibres had lost their neurilemma sheath (Photo 4), an intense lesion was found, which by a low power seemed to consist of large irregular masses arranged in rows. But with a higher power the changes were seen to be of different types. In some fibres there were series of large irregular masses similar to those which are characteristic of Wallerian degeneration; in other fibres the reaction assumed the form of black sinuous threads (see Photos 1 and 3); in some, round or oval globules of various sizes were seen, and some fibres showed no Marchi reaction at all.

These various types of degeneration indicate different degrees of injury to

the fibres. We are thus able to explain the fact that the degeneration was continued along many of the fibres of the descending fifth root, even as far as the first cervical segment (Photo 6). In the case of these fibres there must have been a necrosis of the axis-cylinder, as well as of the myelin sheath, immediately inside the point where the fibres lost their neurilemma sheath, which would produce a Wallerian degeneration in the portion of the fibres beyond the point of injury. In the descending root, however, there were many fibres which showed no degeneration, and these would correspond to the unaffected fibres, which were seen in the first part of the intracranial portion of the fifth nerve, and also to those fibres which exhibited the slighter degrees of change.

In the right fifth nerve round and oval globules were present, but there was no evidence of any necrosis such as was met with in the left nerve. Corresponding to this there was an absence of degeneration amongst the fibres of the right descending root.

Sixth Nerve.—In both sixth nerves there was considerable degeneration demonstrated by the Marchi method. Here the thread-like form was more in evidence, accompanied by many round and oval globules.

Seventh and Eighth Nerves.—In these nerves very little degeneration was found.

Ninth and Tenth Nerves.—On the left side there were many affected fibres in which the degeneration could be traced from near the periphery of the medulla oblongata to the fasciculus solitarius. In the right nerves there was little change.

Eleventh Nerve.—The right nerve in this case showed a marked lesion, which could be followed from the periphery to its nucleus.

Twelfth Nerve.—There was practically no lesion of the fibres of the twelfth nerves.

In this case it was evident, from the appearances found, that an acute degenerative process had supervened on a chronic morbid condition. In the ninth and tenth nerves especially there was evidence that a slow atrophy had taken place, particularly near the border of the medulla oblongata. But in the left fifth nerve, and to a less extent in the left fourth and both sixth nerves, the increase in the amount of the Marchi reaction and the presence of the large, irregular masses, and the fact that the degeneration in the fifth could be followed from the edge of the pons along the fibres of the descending root of the nerve to the first cervical segment, indicated an acute degenerative process.

CASE 2.—Melancholia. At the post-mortem examination a tumour was found in the anterior part of the left lobe of the cerebellum; there was also an acute basal meningitis, which was more marked on the left side of the pons and medulla. In this case the third nerves were not examined.

Fourth Nerve.—Both fourth nerves showed some degeneration which could be followed from the point where the fibres pierced the pia mater to the nucleus of origin. Most of the Marchi reaction was seen in the form of round or oval droplets, but the thread-like type was frequently present.

Fifth Nerve.—The maximum intensity of the meningitic process was at the level of the left middle cerebellar peduncle, and the cranial nerves which were nearest to this region showed most degeneration.

The fifth nerves were both affected, but in the left nerve most of the fibres showed an acute degenerative reaction. This was seen chiefly in the form of round or oval globules of various sizes ; but with this there was also some thread-like degeneration, and in some fibres the affected myelin lay in larger and more irregular masses.

Some degeneration could be traced to the motor nucleus.

In this case, however, although there was such an intense degeneration in the fibres of the left nerve, only a small number of affected fibres were present in the descending fifth root.

Sixth Nerve.—Some Marchi reaction, in the form of round globules, was present in both sixth nerves.

Seventh and Eighth Nerves.—The seventh and eighth nerves on the left side showed considerable degeneration, but the right nerves were affected to only a slight degree.

On the left side the degeneration could be followed from the periphery of the pons to the nucleus in both nerves. In the seventh nerve there was some thread-like degeneration, associated near the periphery with some larger irregular masses ; but in the other parts of this nerve and in the eighth the reaction consisted chiefly of round or oval globules of various sizes.

Ninth, Tenth, and Twelfth Nerves.—In all these nerves, globules of Marchi reaction were present. In the left tenth nerve the degeneration could be traced from the periphery around the fasciculus solitarius to the nucleus ambiguus.

In this case the most important fact to be observed is that the degree of the lesion varied with the intensity of the meningitic process. This was most marked at the level of the middle cerebellar peduncle, especially on the left side, and it was in this region that the cranial nerves were most affected ; in fact, those connected with the lower portion of the medulla, where the meningitic changes were scanty, exhibited only a slight degree of degeneration. The lesion of the left fifth nerve was very acute, as shown by the character of the Marchi reaction, but there were few degenerated fibres in the descending fifth root ; there can be little doubt, however, that if the process had lasted a little longer, necrosis of the axis-cylinders and myelin sheath would have been produced, and there would have followed a Wallerian

degeneration of the fibres extending throughout the descending root.

At the same time it is interesting to find that the lesions in the fibres began at the same point, viz. immediately they had lost their neurilemma sheath, and that they were of the same character, as in cases where the septic focus was extra-cranial (see *infra*).

CASE 3.—General paralysis. Gait markedly ataxic.

Cranial Nerves.—Third and Fourth Nerves.—Some degeneration present in the form of round droplets.

Fifth Nerve.—Both fifth nerves affected to a considerable extent. Descending root nil.

Sixth Nerve.—Considerable degeneration.

Seventh and Eighth Nerves.—Slight degeneration.

Tenth and Twelfth Nerves.—Very slight degeneration. There was considerable degeneration in both superior cerebellar peduncles.

CASE 4.—Melancholia. Cause of death—General tuberculosis. Suppuration of the mastoid cells on both sides.

Cranial Nerves.—Third Nerve.—Some degeneration.

Fourth Nerve.—Considerable degeneration.

Fifth, Sixth, Seventh, and Eighth Nerves.—Much degeneration in all these nerves on both sides. Threadlike, round, and large irregular types present.

CASE 5.—General paralysis.

Cranial Nerves.—Third and Fourth Nerves.—Very little degeneration.

Fifth Nerve.—Only one fifth nerve showed much degeneration.

Sixth Nerve.—Maximum degeneration. Both nerves affected.

Seventh Nerve.—Considerable degeneration on both sides.

Eighth, Ninth, and Tenth Nerves.—Little degeneration.

Twelfth Nerve.—Some degeneration on both sides.

CASE 6.—General paralysis.

Cranial Nerves.—Third and Fourth Nerves.—Slight degeneration.

Fifth Nerve.—Considerable degeneration of the peripheral type, *i.e.* the degeneration was found only in the peripheral portion of the myelin sheath, the rest of the fibre showed no change.

Sixth and Seventh Nerves.—Considerable degeneration in the form of round globules.

Eighth Nerve.—Some degeneration of the peripheral type.

Tenth and Twelfth Nerves.—Very little degeneration.

CASE 7.—General paralysis.

Third and Fourth Nerves.—Practically no degeneration.

Fifth and Sixth Nerves.—Considerable degeneration on both sides.

Seventh and Eighth Nerves.—Slight degeneration.

Ninth, Tenth, and Twelfth Nerves.—Practically no degeneration.

CASE 8.—General paralysis.

Necrosis of skin over the right shoulder, muscles bare, pus burrowing amongst muscles, abscesses in the right trapezius. Very little degeneration in any cranial nerve excepting the eleventh on the right side.

CASE 9.—General paralysis.

Some changes in the *sixth, seventh, and eighth cranial nerves.* Practically nil in the others.

CASE 10.—General paralysis.

There was an acute degeneration in *one fifth nerve.* Practically nil in the other cranial nerves.

In this series of ten cases in which the cranial nerves were examined there are a few points to which we wish to draw attention.

The series included eight cases of general paralysis, one of tumour of the left lobe of the cerebellum in which an acute basal meningitis was the ultimate cause of death, and one case of general tuberculosis. In the last-mentioned and in one general paralytic a septic process was present during life which seemed to have a direct causal relation with the lesion found in the cranial nerves. In the case dying of general tuberculosis this consisted of chronic middle ear and mastoid disease, and the maximum lesion was found in the seventh and eighth nerves, although there was considerable change in the sixth, fifth, and fourth nerves. In the general paralytic there was necrosis of the skin over the right shoulder, with abscesses in the deep muscles of the neck, and especially in the trapezius. The right eleventh nerve showed considerable degeneration, but in the others there was little to be found.

In the remaining cases, in which no septic focus was found, the nerves connected with the pons, fifth, sixth, seventh, and eighth were more frequently and more seriously affected than those connected with the medulla oblongata. Again, the lesion commenced where the fibres lost their neurilemma sheath, and was of precisely the same character in motor and sensory nerves, and in no nerve was there any recognisable change in its extra-pontine portion.

The next point to be considered is the character of the lesion, as exhibited by the Marchi method. All degrees of degeneration were seen. The most intense was that in which there had been a necrosis of the axis-cylinder and of the myelin sheath

in the left fifth nerve of Case 1, with consequent Wallerian degeneration, which could be followed in the descending fifth root to the first cervical segment of the spinal cord.

The other forms in which the degeneration was seen were (*a*) the slender moniliform thread, (*b*) the peripheral type in which the portion of the myelin sheath farthest from the axis-cylinder was affected and the remainder of the fibre showed no change, and (*c*) the type which consisted of small, scattered, round or oval globules. As to the significance of these various types it is impossible at present to speak with certainty, but in all probability they depend upon the intensity of the degenerative process. It should be mentioned that even in the most acute cases, side by side with the fibres showing signs of intense lesion, lay others in which no sign of degeneration was demonstrated by the Marchi method.

The last point to be mentioned is, that in several of the chronic cases the evidences of degeneration were most marked at a little distance inwards from the periphery of the pons or medulla.

This condition is present to a certain extent in most chronic cases, and was well seen in the tenth nerve of Case 1, who was a general paralytic of three years' standing. Further examination of this condition, by Medea's modification of Mann's method (9), demonstrated that although the axis-cylinders could be traced from the point where they pierced the pia mater, no myelin was present around them for some distance inwards. Evidently in these chronic cases a slow atrophy of the myelin takes place in the portion of the fibres most exposed to the attack of some weak toxin which approaches along the perineural sheath of the nerve. This may proceed until the axis-cylinder is laid bare, and therefore, as all the myelin in this peripheral portion of the fibre has disappeared, there is nothing left to give a Marchi reaction.

Toxic Lesions of the Spinal Cord and Cranial Nerves produced Experimentally.

At the suggestion of Professor Lorrain Smith, in whose laboratory the experiments have been conducted, we adopted the following method. Celloidin capsules were filled with a broth culture of a certain organism, then sealed and laid along-

side the exposed sciatic nerve, after which the gluteal muscles were stitched in place and the wound closed. As toxins are known to diffuse through the wall of these capsules, we assumed that they would find their way into the ascending lymph stream of the nerves and so to the cord, where their presence would be demonstrated by the ensuing degeneration. The organisms we first employed were staphylococcus pyogenes aureus, bacillus pyocyaneus, Gaertner's bacillus, and the bacillus coli; later we used the organisms taken from cases of acute colitis. We commenced our experiments at the beginning of 1905 and at first used organisms whose virulence had not been raised in any way. We found, however, that in order to obtain definite lesions it was necessary to do so, and further, that it was advantageous to renew the capsule from time to time in order to maintain the supply of toxin. We purposely ignored at this stage the question of the predilection of certain organisms for the nervous system, and kept strictly to our original intention of confirming our own views and those of others on the direction of the lymph stream in the spinal roots, and the possibility of cord infection by this channel.

Five successful experiments have been examined by the Marchi method; three on the cord and two on the pons and medulla. In the latter the capsule with its bacteria was implanted under the skin of the cheek, and the result of these two experiments has been to demonstrate certain special changes which occur in the intramedullary portion of nerves, especially motor nerves, in toxic conditions. The pons and medulla were cut in serial section in their entirety, and we eliminated as far as is possible the artefacts of injury incidental to the autopsy, by merely removing the bony covering and hardening *in situ* for one week in formalin 10%. Later the tissue was immersed in 2% potassium bichromate before treatment with the Marchi method. Control experiments with capsules containing sterile broth were also performed. These yielded negative results.

In the following description it has been necessary to go into detail, especially with regard to the cranial nerves, as in each of these the degeneration varied in intensity as well as in type, for reasons at present difficult to understand.

Rabbit 1.—Capsule under Skin of Cheek.—Third Nerve.—This nerve was degenerated in its entire intramedullary course from its point of emergence back to the nucleus of origin, towards which, however, the changes were less

marked. The myelin sheath showed as long, thin, black threads, with moniliform swellings at intervals, marking out the path of the nerve in parallel or sinuous lines (Photo 1).

Fourth Nerve.—The degeneration here was much less than in the third nerve. There were sinuous filaments of altered myelin, but these were short, and situated only at the point of emergence. The degeneration rapidly decreased from below upwards, and at the nucleus of origin there was no appreciable change.

Fifth Nerve.—There was a high degree of alteration in both sensory and motor portions. In the latter, the degeneration took the form of short, thin, black filaments with moniliform swellings mixed with swollen globules which could be traced into the main nucleus of origin, and for a short distance upwards along the mesencephalic root.

In the former, the degeneration was of the same type but greater in degree, and extended in the descending root as far as the level of the seventh nucleus.

Sixth Nerve.—There was a higher degree of degeneration in this nerve than in any other, and the morbid appearances were similar to those of the third nerve, but somewhat more advanced. The maximum of degeneration was found in the anterior third of the nerve between a point just posterior to the corpus trapezoides and the margin of the pons. In this portion the myelin was fused into short and long masses of somewhat sinuous outline ; some were completely isolated, ending in swollen bulbs, others were continued as fine black threads (Photo 2). Scattered at intervals there were many isolated finer threads and also polymorphic globules. In the middle third of the nerve these appearances were replaced by parallel sinuous rows of fine black filaments with moniliform swellings, resembling precisely the degeneration in the third nerve (Photo 3). Towards the nucleus of origin these filaments of morbid myelin became finer and fewer in number, but were easily traced curving round the ascending portion of the seventh nerve and ultimately gaining the floor of the fourth ventricle.

Seventh Nerve.—The degeneration here was less than in the third, fifth, or sixth nerves, and approximated rather to that in the fourth. Its commencement at the point where the neurilemma sheath left the fibres was easily demonstrable, and it steadily decreased centralwards ; in fact towards the nucleus there were only traces of altered myelin. The degeneration in the distal portion and ascending limb was mostly of the fragmentary type, with a few examples of filiform myelin change.

Eighth Nerve.—The degeneration was slight and only recognisable in the cochlear division in the form of a few black threads with moniliform swellings ending abruptly lateral to the restiform body.

Ninth, Tenth, and Eleventh Nerves.—The degeneration in these was so insignificant as to be negligible ; while in the *twelfth* only a few blackened myelin threads were visible amongst the nerve strands.

Rabbit 2.—Capsule under Skin of Cheek.—Third Nerve.—The degeneration was very slight. Along the course of the nerve there were flakes of altered myelin and short rows of globules ; fine black threads were rare.

Fourth Nerve.—Exactly as in the third.

Fifth Nerve: (A) Motor Portion.—Here the degeneration was of the same

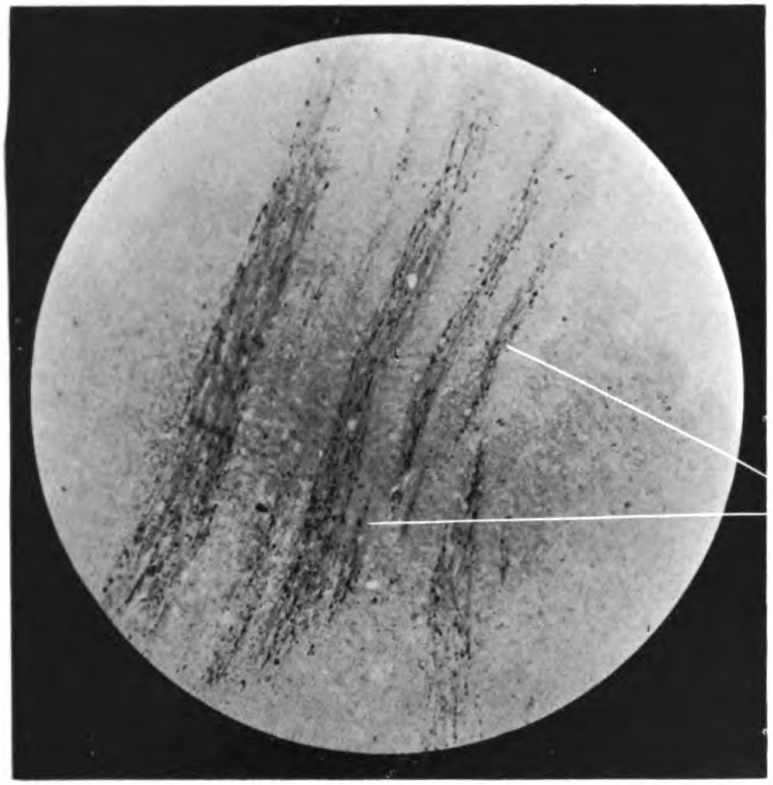

PHOTO 1.—Expt. 1. 3rd cranial nerve. Fine beaded myelin threads.

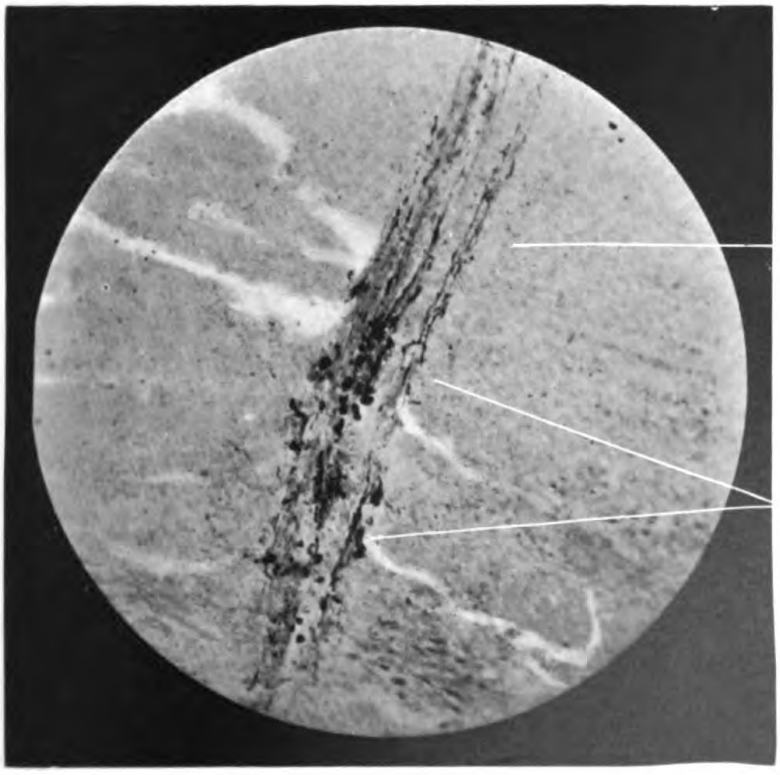

PHOTO 2.—Expt. 1. Anterior third of 6th cranial nerve. 1. Distal portion showing swollen globules of degenerated myelin. 2. More central portion showing black myelin threads.

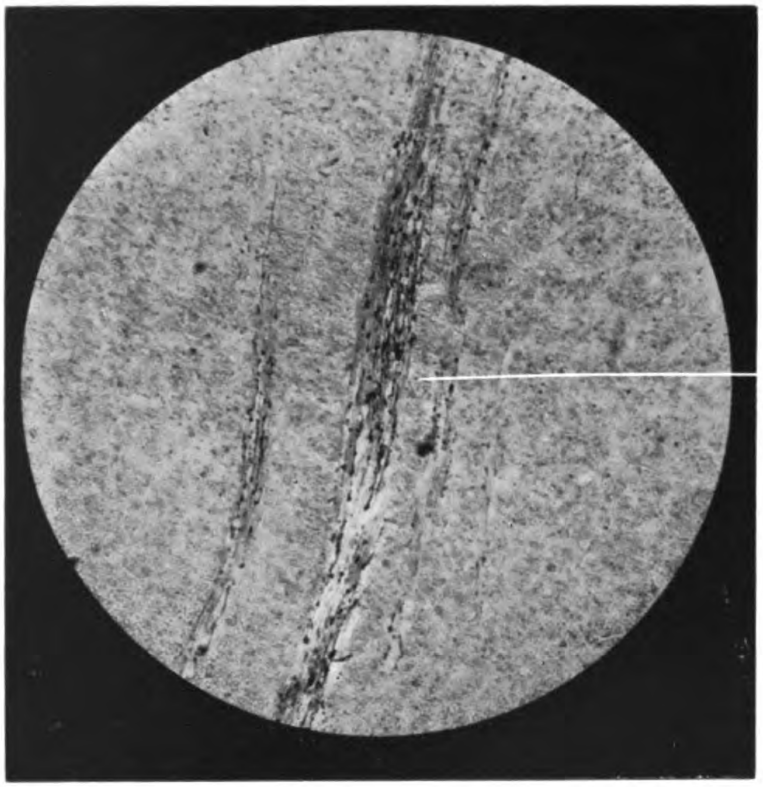

PHOTO 3.—Expt. 1. Middle third of 6th cranial nerve. 1. Sinuous rows of fine black filaments with moniliform swellings.

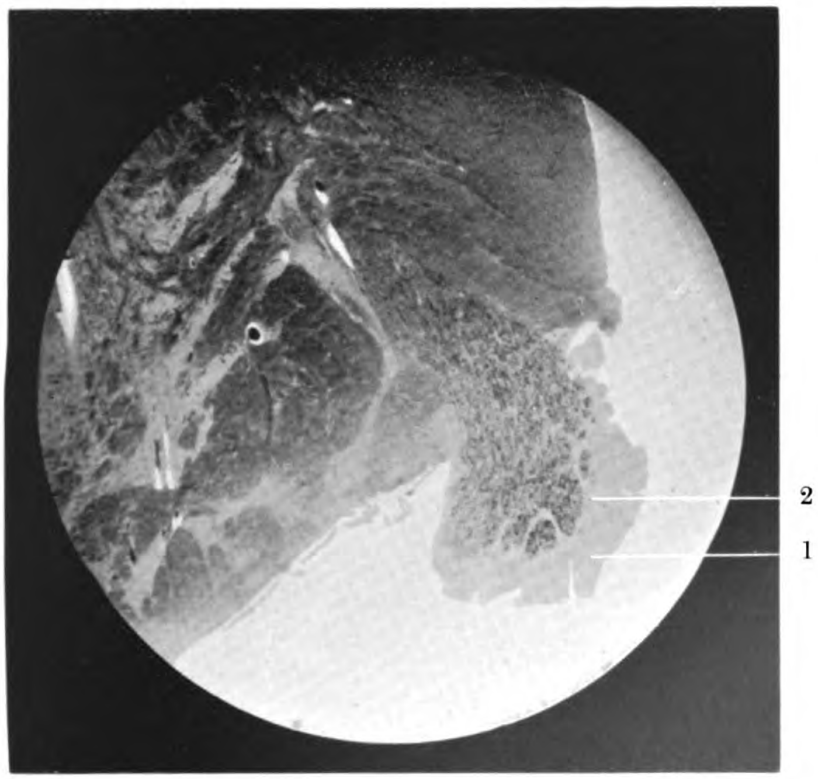

PHOTO 4.—Case 1. General Paralysis. 5th cranial nerve. 1. Extra-medullary portion normal. 2. Degeneration commencing at the point where neurilemma is lost (intra-medullary).

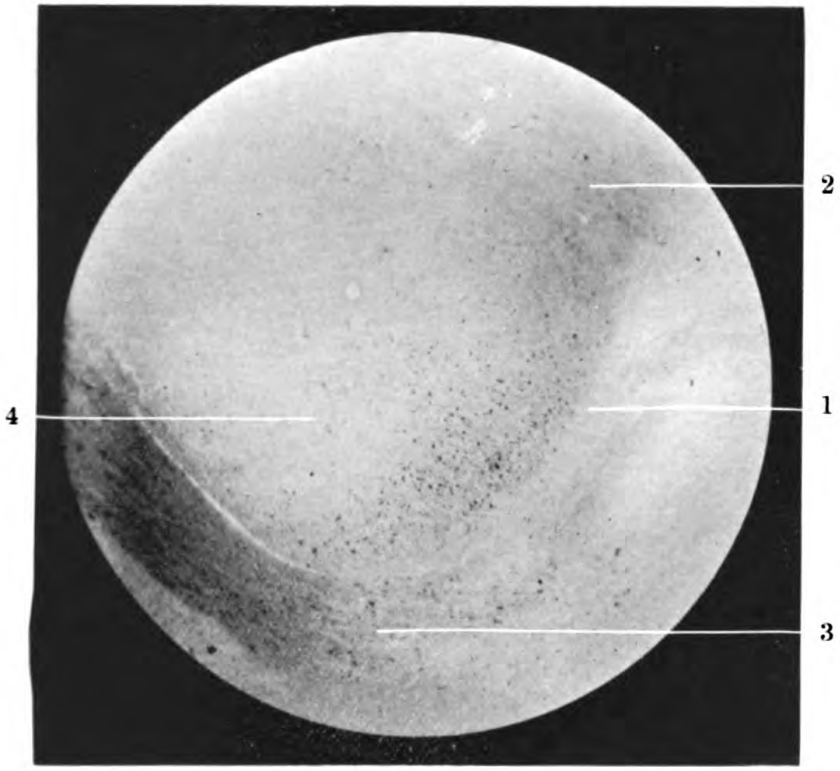

PHOTO 5.—Expt. 2. 1. Ascending root of 5th cranial nerve, containing many degenerated fibres cut in transverse section. 2. 8th nerve. 3. 7th nerve. 4. 5th nucleus.

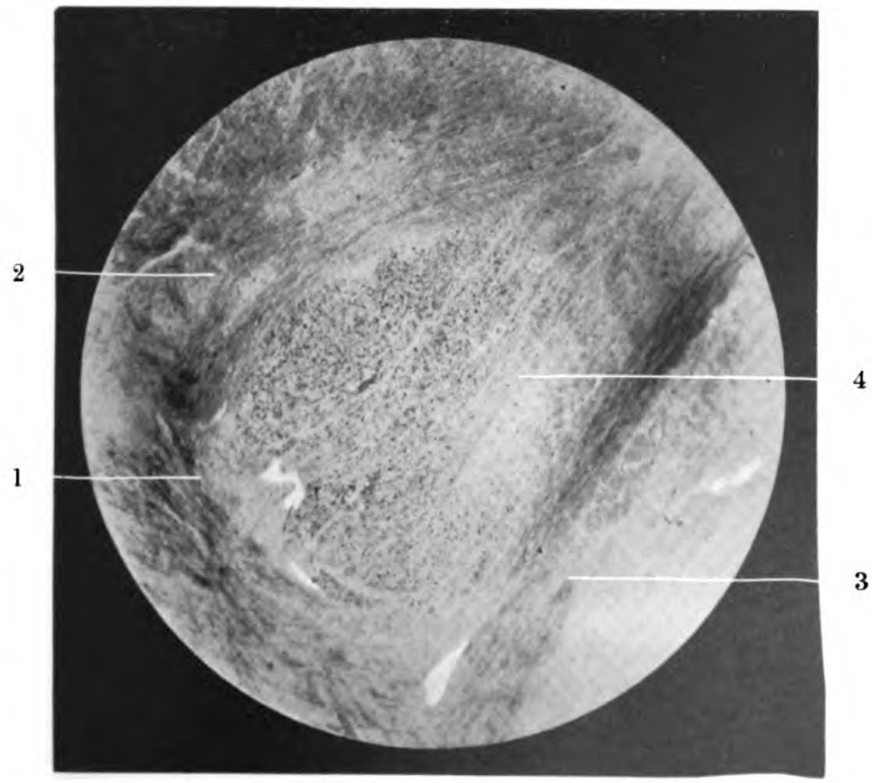

PHOTO 6.—Case 1. General Paralysis. 1. Descending root of 5th cranial nerve, containing many degenerated fibres cut in transverse section. 2. 8th nerve. 3. 7th nerve. 4. 5th nucleus.

type as in the third nerve, but a little more marked. It commenced as soon as the fibres lost the neurilemma and could be traced into the main nucleus of origin, and for a short distance up the mesencephalic root. (*B*) *Sensory Portion.*—The degeneration in this root was very intense, especially at and immediately inside the point where the fibres lost the neurilemma sheath. The altered myelin globules, some small, some large, lay end to end in rows. Frequently one met as many as ten small globules so arranged, and occasionally also fine myelin threads with moniliform swellings in parts disintegrating into fine black granules. In appearance the degeneration coincided with that termed Wallerian, and was traced in the descending root as far as the lower part of the medulla (Photo 5).

Sixth Nerve.—There was more degeneration here than in the third and fourth nerves, and it attained its maximum in the anterior third just inside the corpus trapezoides. The myelin was fused into globules of varying size and shades of osmic acid staining, some being dark brown, others black; but the blackening was never so striking, nor the myelin fusion so complete as in the preceding case. Even portions of the same globule varied in shade, and the large majority were vacuolated. Many sinuous threads were present, black or brown in colour, which often formed connecting bands between adjacent fusiform masses of degenerated myelin, or others separated by a considerable interval (Photo 7). Half way towards the nucleus these threads were still present, but absent in the posterior third of the nerve and at the nucleus itself.

Seventh Nerve.—The degeneration was slightly greater than in the third, fourth, and motor root of the fifth nerve, and was practically confined to the distal intramedullary portion, where the nerve emerges. Here the myelin had disintegrated into small and large globules arranged in rows; there were a few examples of the thread form of degeneration.

Eighth Nerve.—The degeneration was very slight, and in the other nerves, ninth, tenth, eleventh, and twelfth, there was no change worthy of note.

It is necessary to preface the description of the experiments on the cord by stating that in the first, in addition to placing a capsule under the sciatic nerve, one was implanted under the muscles covering the upper dorsal region. The wound here became septic twenty-seven days after the operation, owing to rupture of the capsule, and the pus invaded the cervical region on the right side before the accident was discovered, when the animal was immediately killed.

Rabbit 3.—In the lowest segments of the cord no degeneration was present. In the lumbar enlargement, however, the osmic acid method yielded a very definite positive reaction. The degeneration of the exogenous system commenced immediately the fibres lost their neurilemma sheath, involved the root entry zone and the whole of the posterior columns with the exception of a small area behind the commissure, and the posterior periphery of the cord. There was considerable degeneration of the collaterals, most evident in those

running through the antero-internal angle of Rolando's substance into the posterior grey horns; while those passing towards the anterior cornua showed much less alteration (Photo 11). From the lumbar region upwards through the lower dorsal cord there was a steady diminution in the number of degenerated fibres, those remaining being scattered around the median septum; but in the upper dorsal region degeneration again appeared in the root entry zones, especially in that of the right side. The degeneration here, however, had not the systematic character of that in the lumbar enlargement. On reaching the cervical region there was a marked increase in the number of degenerated fibres, these almost completely filling the posterior columns and leaving free the same areas as noted in the lumbar cord. The collaterals leaving the root entry zones were degenerated, but not further forwards than the level of the commissure.

The Marchi reaction in this case was, in the large majority of instances, of the usual type seen in transverse section, each degenerated fibre showing as a solid black globule; but occasionally appearances indicating partial degeneration of the myelin were met with. For example, Photo 8 shows a fibre in which the black degenerated outer and periaxial portions of the myelin are separated by a clear ring in which the osmic acid reaction is negative. This middle area in many other examples stained dark grey or brown, the colour gradually merging into the black on either side. Occasionally, too, the peripheral part of the myelin alone stained. In the root entry zones sometimes there were solid, elongated segments of degenerated myelin (Photo 9). In longitudinal section the degeneration showed as long parallel lines of myelin droplets.

Rabbit 4.—The description of the degeneration in the above experiment holds good for this one with this exception, that here the morbid changes were not so advanced; so that partial degeneration was very frequently seen (Photo 8). The degeneration diminished rapidly on passing from the lumbar to the dorsal region.

Rabbit 5.—Of the three cords this was least affected. The distribution of the lesion in the lumbar enlargement was the same, but the affection of the collaterals was much less noticeable. There were many more examples of partial degeneration, sometimes only three-fourths of the myelin circumference being blackened, leaving the remaining fourth merely darkened. There were many fibres in which the myelin was degenerated only round the axis-cylinder, the outer myelin ring remaining normal; in others this also was blackened, leaving a middle clear zone. The uniform degeneration of the whole diameter of the fibre was not found nearly so frequently as in the other two cords.

The extra-medullary part of the posterior roots and of the cranial nerves was examined by Marchi's method. No degenerative changes were found. In the cord of one case (Experiment 3) there was some degeneration of the lateral columns in the vicinity of the posterior horns in addition to that already noted in the posterior columns; but in the pons (Experiments 1 and 2) the degeneration kept strictly to the course of the nerves with one exception, viz. in Experiment 1, in which there was some degeneration of the fibres of the corpus trapezoides for a short distance on either side of the sixth nerve.

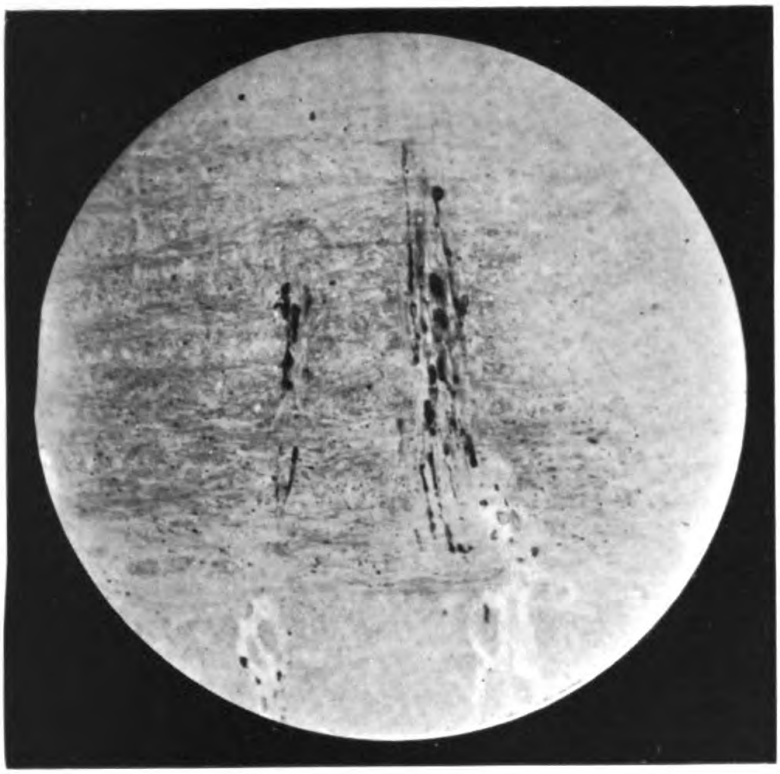

PHOTO 7.—Expt. 2. 6th cranial nerve.

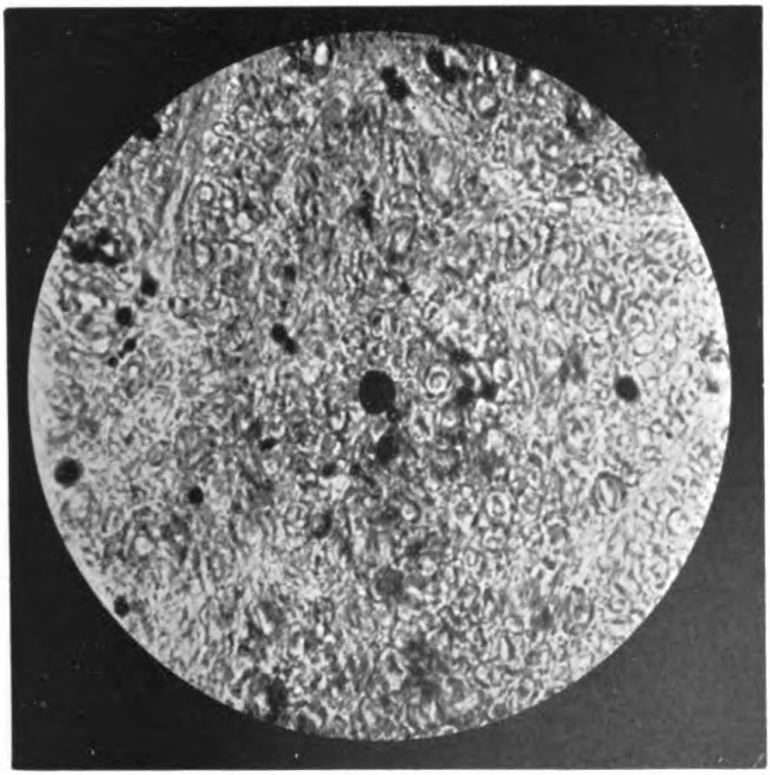

PHOTO 8.—Expt. 3. Degeneration of inner and outer parts of fibre.

PHOTO 9.—Expl. 3. Elongated segment of degenerated myelin.

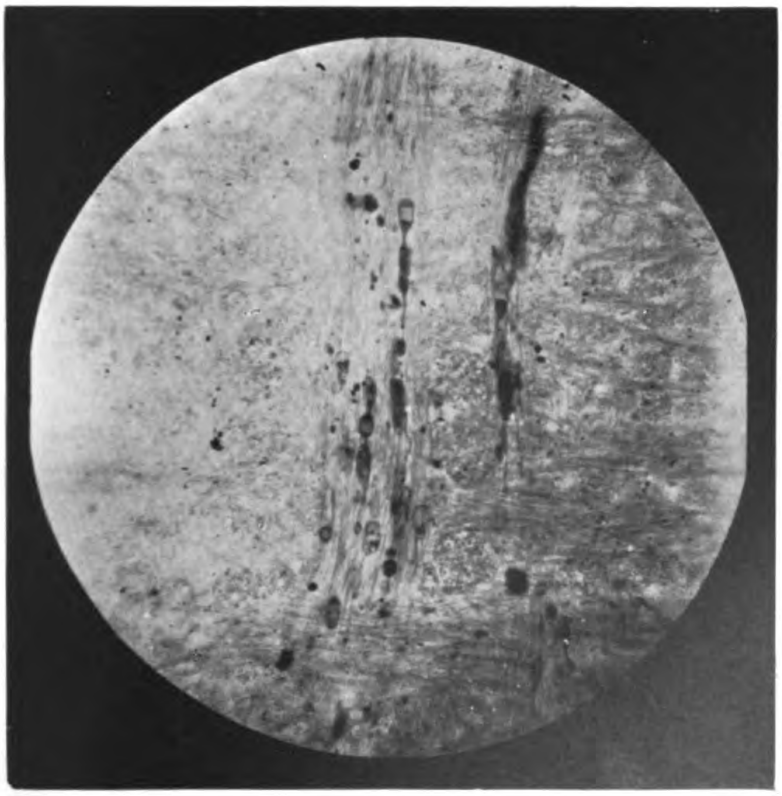

PHOTO 10.—Expt. 2. 6th cranial nerve; darkening of myelin sheath with vacuolation.

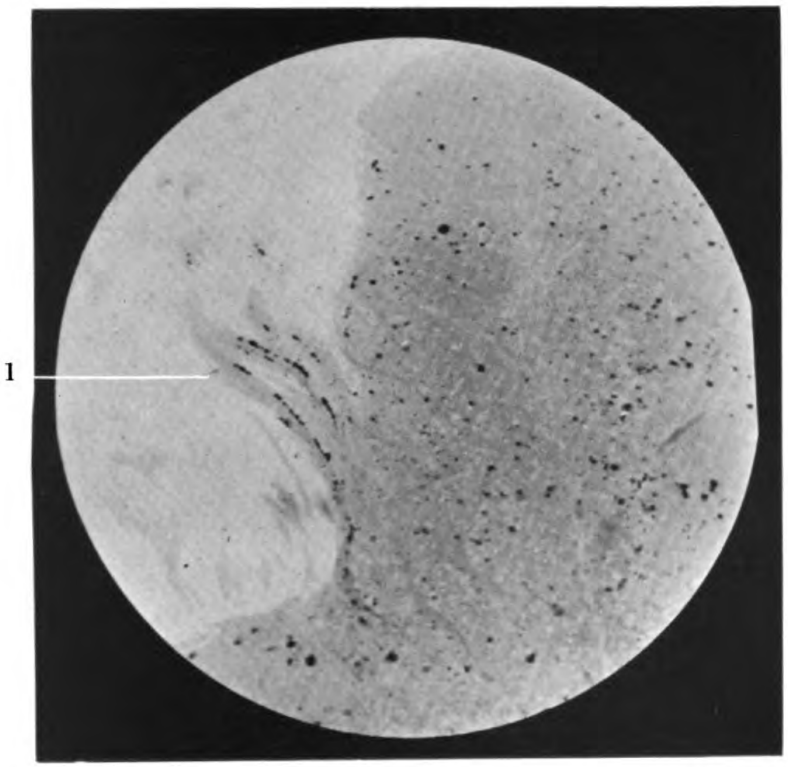

PHOTO 11.—Expt. 3. Degeneration of posterior columns and of the collaterals passing into the posterior grey horn.

In discussing the nature of the degenerations above described it will be advantageous to commence with a motor cranial nerve. Taking the sixth as an example, and comparing the lesions in Experiments 1 and 2, we can follow the degeneration from an early to an advanced stage. The initial change (Experiment 2) is a darkening of the myelin sheath with swelling and vacuolation (Photo 10), passing on to the formation of the thick fusiform masses and thin, elongated, moniliform structures which characterise the later degeneration in Experiment 1. The distal part of the intramedullary portion of the nerve suffers first (Experiment 2), followed by involvement of its whole length back as far as the nucleus of origin (Experiment 1). Meanwhile the extramedullary portion remains intact. The lesion, therefore, is not Wallerian, such as was present in the fifth descending root (Experiment 2), for, commencing distally, it extended centralwards and gradually diminished in that direction as the toxin lost potency in its progress along the nerve. Both in appearance and in its mode of extension this degeneration is typical of that found in toxic conditions, and strongly resembles in character the variety known as "segmentary necrosis."[1] The black fusiform masses and long sinuous threads point clearly to an acute necrotic or liquefactive change in the medullary sheath. As to whether the axis-cylinder was involved in the process we cannot say without supplementing the Marchi method by other stains.

In the other cranial nerves and in the cord there is abundant additional evidence of primary myelin degeneration of varying degree, well exemplified in the transverse sections of the cord. All stages of alteration are present, from that confined either to the neighbourhood of the axis-cylinder or to the periphery of the myelin ring (Photo 8), to a more advanced phase where the medullated sheath is seen as a solid black globule. Where these globules are present in sufficient number and can be traced in a centrifugal direction for some distance, one must admit involvement of the axis-cylinder with Wallerian degeneration, *e.g.* in the fifth descending root of Experiment 2 and of Case 1, and in the root entry zones and collaterals of the cord in Experiment 3. In toxic conditions, therefore, primary myelin change is the forerunner of secondary degeneration whose onset must be deter-

[1] Cornil et Ranvier. Man. D'Histol. Pathol., T. 3, pp. 688, 715.

mined either by the intensity of the toxin or by the duration of its action. Low virulence will account for the slow myelin atrophy and disintegration so often met with in many nervous affections, and we need only mention in this connection general paralysis and tabes dorsalis, in which much myelin degeneration can exist with comparative integrity of the axis-cylinders. But there comes a time when this structure's resistance is overcome in the natural course of events or by a rise in virulence of the exciting toxin, and secondary degeneration ensues. In general paralysis of the acutely delirious type we have seen acute Wallerian degeneration in the posterior columns of the cord and in the cranial nerves, which could only be produced by some very potent toxin ; while in other chronic cases there have been changes ranging from simple atrophy to more advanced phases of degeneration. It is this variation in toxicity which constitutes the difference between the slow and rapid types of the above mentioned progressive lesions.

Conclusions.

(1) In peripheral nerves, spinal roots, and cranial nerves there is a constant stream of lymph ascending towards the central nervous system whose main current lies in the inner meshes or lymph spaces of the fibrous perineural sheath. Toxins reach the spinal cord and brain by this channel ; and although they spread to some extent in the lymph spaces of the pia-arachnoid, and so affect structures at a distance from their point of entry, for the most part they pass, in the main current of the lymph, along the nerve roots into the substance of the central nervous system. Here they apparently follow the nerve paths of the affected roots, and show little tendency to diffuse amongst the neighbouring fibres (Photos 2 and 3). (2) In their extramedullary portion these nerves are protected from the influence of the toxins by the vital action of the neurilemma sheath, but on losing this in their intramedullary part they at once undergo degeneration. (3) The first change is a primary degeneration of the myelin ; the axis-cylinders and nerve cells are evidently affected later.

Since writing the above, we have met with and are now investigating a case of general paralysis which bears out to a very marked degree the experimental and clinical evidence given

above. At the autopsy the following morbid appearances were found, suggesting the presence of some infective condition, *e.g.* acute catarrh and ulceration of the large intestine with hæmorrhages, especially of its lower portion, chronic thickening of the mucous membrane of the bladder, with an acute cystitis and very offensive blood-stained urine. In addition we should mention the presence of a septic condition of the right knee-joint which had existed for two weeks previous to death. Cultures were made from the cerebro-spinal fluid of the lateral ventricles and of the lumbar region, and yielded an abundant growth of a rod-shaped bacillus which often assumed a long thread-like form. In sections of the right sciatic nerve we failed to find any organisms, but found them abundantly in the following regions, viz.: in the internal layers of the perineural sheath of the posterior and anterior lumbar roots, amongst the fibres of the posterior root entry zone, in the pia-arachnoid around both sides of the cord, and in the septa passing into the cord where they were most numerous at the periphery, amongst the anterior root fibres, and a few in the central canal and surrounding tissues. In the spinal roots the organisms also followed the track of the vessels passing into the centre of the nerves. In both roots and cord there were many in the adventitial lymph spaces of the vessels, but nowhere did we observe a single organism within the lumen. The vessels were dilated, and there was cellular infiltration of their walls. In the cord the organisms were found to be limited to those areas which we have shown to be affected as a result of toxic absorption (8). This is especially well illustrated in comparison with a case of brachial neuritis (10). It will be seen here that the distribution of the myelin degeneration in the posterior root entry zone, the anterior radicular zone, around the pia-arachnoid, and along the septa to the central parts of the white matter, coincides exactly with that of the organisms in the present case.

The presence of the organisms in the adventitial sheath of the vessels and their entire absence within the lumen are facts of very great significance. We would suggest that this infection of the adventitia is secondary to the infection of the lymph circulating amongst the nerve fibres. In support of this view we would mention that it is frequently found, in advanced phases of cord degeneration, that the fatty products in process of granular disintegration and absorption pass into the adventitia

and fill the cells and spaces there until the vessel wall may be stained almost uniformly black with osmic acid. It is possible that this is the true explanation of the vascular changes found in the cord in acute myelitic lesions, and that these should not be considered as being of primary hæmatogenous origin, but as being reactive changes due to a primary lymphogenous infection of the adventitia of the vessel walls.

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ON THE NATURE OF THE "FAISCEAU EN ÉCHARPE" OF FÉRÉ.

By G. ELLIOT SMITH, Cairo.

IN the account of the crus cerebri in Testut's "Traité d'Anatomie humaine" (tome ii., 1900, p. 598), it is stated that the bundles of fibres of which the cerebral peduncles are composed are usually arranged in a parallel manner, but "dans certains cas les faisceaux nerveux obliquent en masse d'arrière en avant et de dedans en dehors, comme si le pédoncule était tordu sur son axe"; and, further, "dans d'autres cas, on voit les fibres les plus internes abandonner brusquement leur situation primitive, croiser à la manière d'une écharpe la face inférieure du pédoncule pour venir occuper son côté externe (fig. 469, 3): ce faisceau oblique, auquel FÉRÉ a donné le nom de *faisceau en écharpe*, est rarement

symétrique." Testut's account then goes on to distinguish this "sling-band" from the *taenia pontis* of HENLE and the *tractus peduncularis transversus* of v. GUDDEN.

In the accompanying drawing of the left lateral aspect of the mesencephalon of a Soudanese negress (Fig. 1), all three bands are present. The "*Faisceau en écharpe*" is labelled "*Fasc. obl. cruris*," a contraction of the descriptive term "*Fasciculus obliquus cruris cerebri*," with which I propose to distinguish the bundle in question. It obviously consists of some of the most lateral fibres of the crus cerebri which dissociate themselves from the rest of that body, and, after passing obliquely downward (caudally) and inward on the surface of the crus, enter the interpeduncular space and plunge vertically downward into the substance of the pons varolii. The rest of its course is shown in the accompanying photograph of a sagittal section of the same brain (Fig. 2) as a very distinct, bow-shaped, white strand along the line of separation of the pontine fibres from the formatio reticularis. In this section the pontine region was cut about one millimetre to the left of the mesial plane, and thus revealed this fasciculus (which is present only on the left side) with extraordinary clearness. Without any further preparation, a vast number of tree-like bundles of fibres were clearly visible passing forward into the pons (Fig. 3), to terminate presumably in the nuclei pontis. No fibres could be detected passing backward into the formatio reticularis.

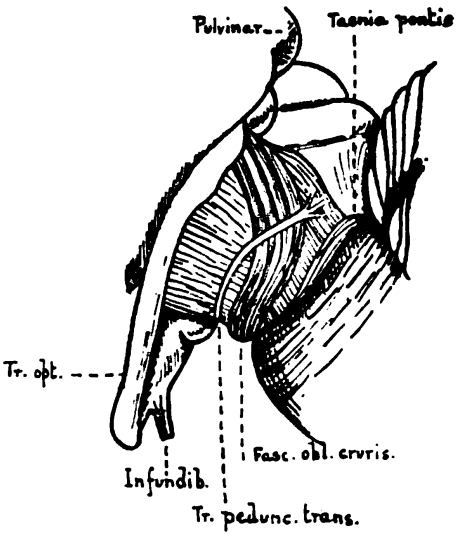

FIG. 1 — The left lateral aspect of the mesencephalon of a Soudanese negress. *Fasc. obl. cruris* is the "*Faisceau en écharpe*" of FÉRÉ.

For the purpose of locating this bundle more accurately, the two halves of the mesially-split pons were put together and cut in transverse section. The *fasciculus obliquus cruris* was thus exposed lying in the formatio reticularis in close apposition to the inner extremity of the mesial fillet (Fig. 4, *lemniscus*),

with which it might easily be confused in studying transverse sections.

The *fasciculus obliquus cruris* is clearly a part of the peduncular system ending in the nuclei pontis. In this particular specimen I was unable to trace the bundle higher than the corpus geniculatum laterale, under which it emerged

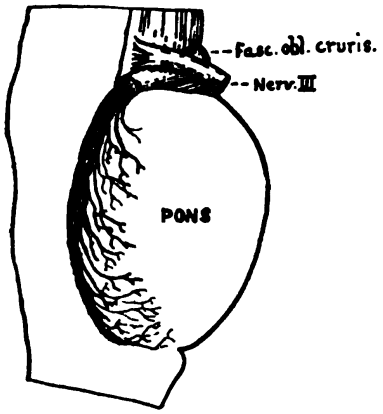

FIG. 3.

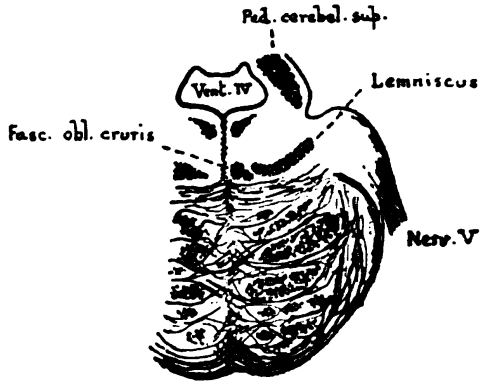

FIG 4.

FIG. 3.—A semi-diagrammatic representation of the fibres exposed in the left half of the pons that was removed from the specimen shown in Fig. 2.

FIG. 4.—Part of a transverse section through the pons shown in the other three figures. The section passes through the insertion of the trigeminal nerve (*Nerv. V.*), and exposes the *fasciculus obliquus cruris* on the left side of the mesial raphé, near the inner end of the lemniscus. The posterior longitudinal bundle is shown but not labelled.

from the cerebrum (Fig. 1); but in other brains that I specially examined for this purpose I could trace the fibres of the extreme lateral part of the crus into the temporal region of the hemisphere. It is most probable that the *fasciculus obliquus* is part of the path connecting the temporal region of the left cerebral hemisphere with the right cerebellar hemisphere.

Taking three other human brains at random, I found this "oblique bundle" in two of them; and in all three cases it occurred on the left side. In one specimen only there was a small bundle on the right side. In none of the specimens examined have I been able to demonstrate its mode of termination with the clearness exhibited in the chance-section shown in Figs. 2 and 3.

The apparent predilection of this bundle for the left side is interesting, especially when it is recalled that the aberrant circumolivary bundle of the pyramidal tract is found only on the

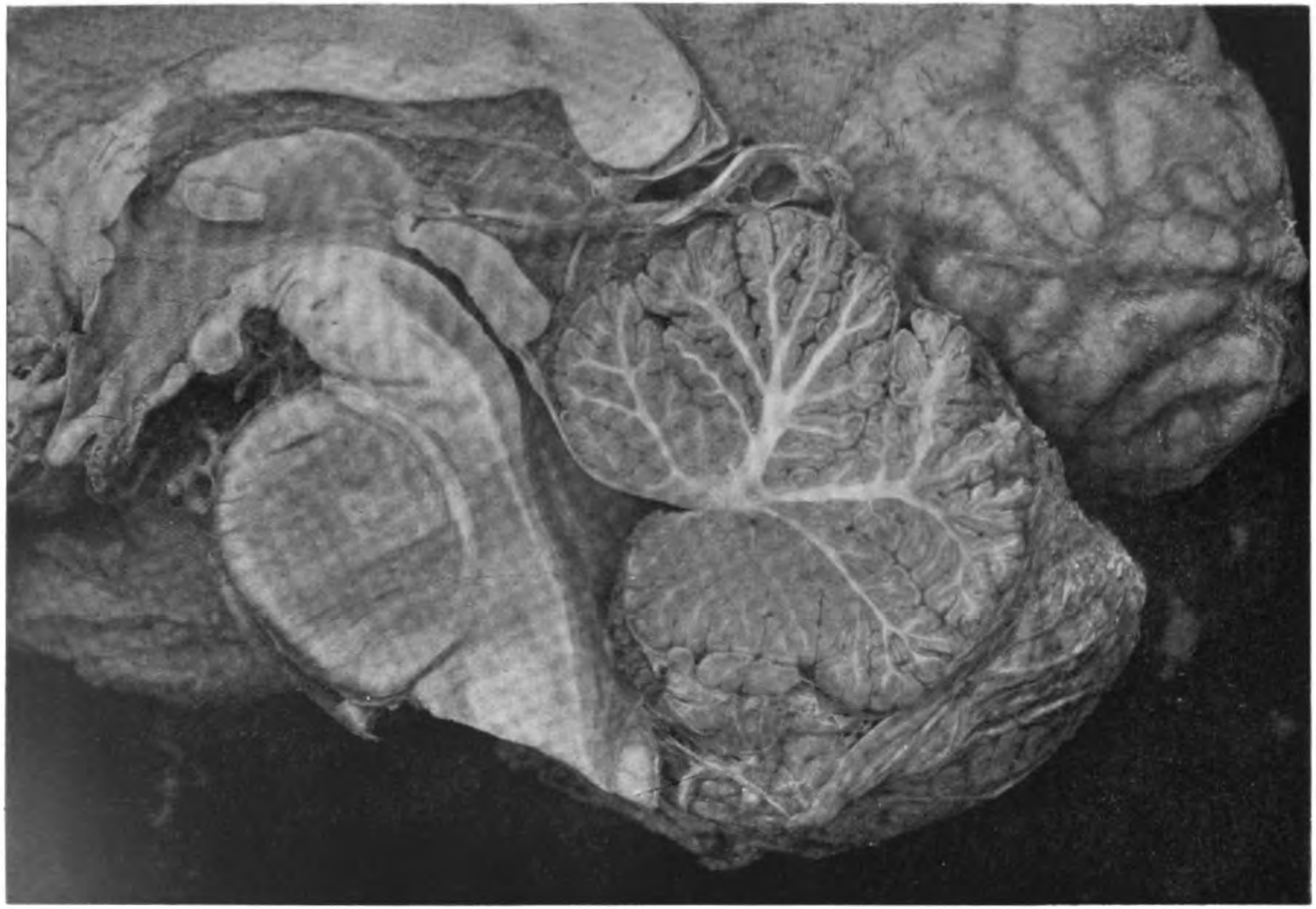

FIG. 2.—Untouched photograph of a sagittal section of the same brain, showing the terminal portion of the *fasciculus obliquus cruris*. This section passes through the pontine region, one millimetre to the left of the mesial plane.

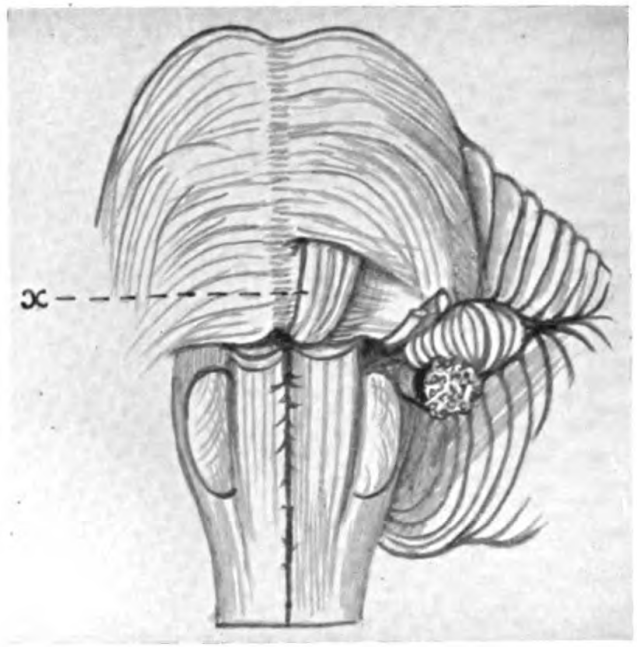

FIG. 1.

left side in the vast majority of cases (see *Review of Neurology and Psychiatry*, vol. ii., No. 5, May 1904, p. 377). Among more than one hundred examples of the latter anomaly I have seen only three cases in which it was present only on the right side. It seems as if there were a distinct tendency for fibres to be crowded out of the left peduncular tract into aberrant courses.

ON ANOTHER FORM OF ANOMALY IN THE CEREBRO-PONTINE TRACT.

By G. ELLIOT SMITH, Cairo.

DURING the week that has elapsed since I posted the account of the aberrant course of a part of the left cerebro-pontine tract for publication in this Journal, I have examined twenty-five brains, and found ten examples of a similar anomalous bundle. In every case it occurred on the *left* side: in one case a smaller bundle of the same nature was found on the right side, and in another case symmetrical aberrant bundles were found.

During the course of the search for the "*faisceau en écharpe*," I have found a most curious anomaly of a serially homologous bundle of fibres, which is represented in the accompanying diagram and photographs.

It was found on the *left* side only. At the junction of the lower and middle thirds of the pons a large vertical band of fibres was seen emerging (Fig. 1, *x*) between the transverse pontine fibres just to the left of the groove for the basilar artery, and passing downward on the surface of the pons. At the lower border of the latter the fibres became collected into a prominent round cord, which plunged horizontally into the substance of the hind-brain. A vertical section through this curious anomaly, about 3 mm. from the mesial plane (Fig. 2), clearly reveals its nature: it is the most superficial of the bundles prolonged downward through the pons from the pedunculus cerebri. In a second vertical sagittal section, about 1 mm. from the mesial plane (Fig. 3), the further course of the bundle can be seen horizontally backward below the pontine fibres, and then upward between the latter and the formatio reticularis, to end, like the "*faisceau en écharpe*," which is also present in this specimen (Fig. 2), in the nuclei pontis.

THE RELATIVE ORDER OF INNERVATION OF CERTAIN MUSCLES OF THE ARM.

By ASHLEY W. MACKINTOSH, M.D., Aberdeen.

It is generally accepted that the deltoid muscle, flexors of the elbow and supinator longus are innervated chiefly from the fifth cervical segment of the cord, and that the intrinsic muscles of the hand receive their nerve supply from the first dorsal and eighth cervical segments. According to most clinical records, the pronators are represented at about the same level of the cord as the triceps, which is generally placed at the sixth or sixth and seventh cervical segments; the extensors of the wrist and long extensors of the fingers are usually grouped together at the level of the seventh cervical segment, somewhat lower than the triceps but higher than the flexors of the wrist and long flexors of the fingers, which are also usually grouped together and placed at the eighth cervical segment.

I have recently had a case of fracture of the cervical spine which appears worthy of record as showing, with almost experimental precision, the relative order of innervation of certain arm muscles. Operation and, later, autopsy, disclosed fracture of the body of the sixth cervical vertebra, with a comminuted fracture of the left lamina of the fifth and the right lamina of the sixth vertebra. There was no complete division of the cord, but microscopical examination showed an almost complete crush at the seventh cervical segment, while the eighth segment had also been greatly compressed. Even macroscopically it was evident that the upper limit of the cord lesion was higher on the left side—there was no obvious implication of the sixth segment on the right side, while the lower part of this segment was clearly involved on the left side. This difference on the two sides was confirmed by microscopical examination: on the right side the grey matter of the sixth segment appeared intact, while on the left side the lower part of the sixth segment had shared in the crush, and even the upper part was greatly distorted and the ganglion cells of the anterior horn were markedly diminished in number and abnormal in appearance. The fifth segment was intact on both sides; no lesion other than the crush, *e.g.* an isolated focus of hæmorrhage, could be found.

Clinically, there was complete and permanent loss of motion and sensation on all parts innervated below the site of lesion: the breathing was entirely diaphragmatic. In view of the site and severity of the lesion, the duration of life appears remarkable: the patient lived 355 days.

The condition of the arms, as regards motor power, was as follows:—

	Right.	*Left.*
Muscles of shoulder, Flexors of elbow, Supinator longus	Good power throughout.	Good power throughout.
Extensors of wrist	Very fair power from the first, which improved.	Practically no power at first, slight power later.
Pronators	No certain power at first, later, fair power.	No power.
Triceps	No power at first, ?? slight power later.	No power.
Extensors of fingers, Flexors of wrist, Flexors of fingers, Intrinsic muscles of hand	No power throughout.	No power throughout.

In view of this distribution of the motor paralysis in the arms and of the situation of the lesion in the cord, this case appears to warrant the following conclusions:—

(1) The extensors of the wrist are represented in the cord at a level higher than the triceps, the former almost certainly at the sixth cervical segment, the latter below this segment.

(2) The pronators are represented below the extensors of the wrist and above the triceps, probably at the sixth and seventh cervical segments.

(3) The extensors of the wrist are represented at a higher level than the extensors of the fingers.

(4) The extensors of the wrist are represented at a higher level than the flexors of the wrist.

Stewart and Turner (*Brain*, 1900, p. 139) record a case of dislocation of the fifth cervical vertebra forward on the sixth, with severe crush of the cord at the upper level of the seventh cervical segment; the distribution of motor paralysis in the arms was very similar to that noted in my case, with this exception, that the flexors as well as the extensors of the wrist remained intact. Stewart concludes that these two groups of muscles are

grouped *together* at a level of the cord higher than the seventh cervical; my case does not confirm this conclusion.

It may be of interest to place in tabular form the conclusions as to the segmental or root supply of the arm muscles arrived at by such writers as Ferrier and Yeo (1) (experimental), Herringham (2) (anatomical), Gowers (3), Thorburn (4), Kocher (5), Starr-Edinger (6), Oppenheim (7), Horsley (8), Purves Stewart (*loc. cit.*).

	Ferrier and Yeo.	Herringham.	Gowers.	Thorburn.	Kocher.	Starr-Edinger.	Oppenheim.	Horsley.	Stewart.
Triceps . .	6, 7, 8	7, 8	6, 7, 8 chiefly 7	6	6	6, 7	6, 7	7	6, 7 chiefly 7
Extensors of wrist . .	5	6, 7	6, 7 chiefly 6	7	7	6, 7	6, 7 chiefly 7	7	6
Long extensors of fingers . .	5	—	6, 7	—	8	6, 7	6, 7 chiefly 7	7	7
Pronators .	6, 8	—	6, 7	6	6	6, 7	6	—	6
Flexors of wrist . .	6, 8	—	7, 8	8	7	7, 8	7	8	6
Long flexors of fingers .	7, 8	—	7, 8	—	8	8	8	8	7

The numbers refer to cervical segments or roots.

Analysing these results from the point of view of the four conclusions drawn from my case, we find that—

(1) Ferrier and Yeo on experimental grounds, and Herringham from anatomical data, place the extensors of the wrist higher than the triceps; Gowers and Stewart arrive at the same conclusion, but most clinical records place the triceps either higher than the extensors of the wrist (Thorburn, Kocher) or at the same level (Starr-Edinger, Oppenheim, Horsley).

(2) The writers quoted are generally agreed in placing the pronators at or about the same level as the triceps.

(3) Most of the writers who specially note the point (Ferrier and Yeo, Gowers, Starr-Edinger, Oppenheim, Horsley) place the extensors of the wrist and long extensors of the fingers together at a common level; Kocher and Stewart place the former at a higher level than the latter, and Campbell Thomson (*Brain*, 1899, p. 136) records a case which strongly supports this view.

(4) Most of the authors quoted (Ferrier and Yeo, Gowers, Thorburn, Starr-Edinger, Oppenheim, Horsley) place the flexors of the wrist below the extensors of the wrist; Kocher and Stewart place them at the same level.

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Abstracts

ANATOMY.

COMPLETE BILATERAL INTERRUPTION OF THE FISSURE OF ROLANDO.

(I92) DAVID WATERSTON, *Journ. of Anat. and Phys.*, Vol. xli., p. 143.

IN this communication is recorded an account of the brain of a newly-born child in which the fissure of Rolando in each hemisphere was interrupted near its centre by a short gyrus connecting the precentral with the postcentral gyrus.

This condition has been met with in some instances in the adult brain, but has not hitherto been proved to be present at birth. AUTHOR'S ABSTRACT.

PSYCHOLOGY.

THE CALL OF SOCRATES. (La Vocation de Socrate. Specimen d'une application de la science psychologique à celle de l'histoire.) PIERRE BOVET, *Arch. de Psychol.*, Jan. 1907, p. 261.

(193)

THIS paper is a clever and plausible attempt to whitewash the Delphic Oracle, and incidentally a reminder of the reservation, somewhat of a disappearing mirage in these days of the predominance of the devotedly documentary school of historians, that the splendidly scrupulous methods of the *École des Chartes* may profit betimes by the application of the accessory sciences of mind to that synthetic interpretation of the scoriæ of tradition which even more than the aggregation of guaranteed facts is (as Polybius pointed out long ago) the highest function of the historian. Paradoxically the thesis it chances to sustain involves the straightening out by one rationalist organon of the twist inflicted by another, and the restoration of credit to an admittedly authentic text which the commentators have felt obliged to explain away.

Agreed as to the historical truth of Plato's chapter recounting the solicitation of the opinion of the Oracle by Cheirephon, both Zeller and Gomperz are also at one in holding that the deliverance merely confirmed in Socrates an already formed conviction of a special mission, and had no direct influence on the awakening of his dialectic genius. Both critics go in the teeth of the text to make the absolute assumption that Socrates was already a well-known personage whose reputation was made. M. Bovet denies the judiciousness of this attitude. Reconstituting the scene on the lines of Monceaux's article in Daremberg and Saglio's *Dictionnaire des Antiquités*, he suggests that whereas Zeller, and especially Gomperz, envisage exclusively the priests of the shrine, cold, calculating, prudent office-holders, concentration of the attention on the Pythia herself transforms the problem into one in the psychology of ecstatics. The transaction is then resolved into a simple case of what in popular parlance is termed "thought reading." It was enough that Cheirephon entered the *adyton* with the desire of obtaining a reply favourable to the friend whose great gifts he was possibly the first to appreciate. Telepathy need not be invoked so long as other hypotheses serve. Muscle reading may be excluded, since the name Socrates is not easily to be divulged by an ideo-motor activity, while there is no record of any mediate or immediate contact between agent and percipient during the interview. But the Danish savants, Hansen and Lehmann, in their researches on involuntary whispering, have shown that, even when the mouth is shut, a word present in the

mind is in many cases whispered unconsciously abroad in tones too low to be audible to the ordinary ear, but demonstrable with the assistance of delicate apparatus. Auditory as well as visual hyperæsthesia sometimes accompanies the hypnotic trance, and in that state the Pythia would be particularly prone to lend a favourable ear to the all but unuttered suggestion of her interlocutor.

Part of the paper deals with the thorny question of the *daimon* of Socrates. M. Bouvet arrives by an independent route at the conclusion expounded in F. W. H. Myers' "Human Personality." He finds an exact parallel in the "Léopold" of Mlle. Smith, a detailed study of which appears in Flournoy's book, "Des Indes à la Planète Mars" (Geneva, 1900), and is there explained as a product of psychological duplicature.

J. ROY TANNAHILL.

THE MECHANISM AND LOCALISATION OF THE PSYCHICAL

(194) **PROCESSES. (Uber den Mechanismus und die Lokalisation der psychischen Vorgänge.)** ERNST JENDRASSIK, *Neurolog. Centralbl.*, März 1 u. 16, Jahrg. xxvi., S. 194 u. 254.

THE author first points out that many of the recent controversies on this subject, as on that of aphasia, are complicated by the assumption of old conceptions about the soul. Terms such as "centre d'idéation," "Assoziationscentrum," "Centrum für geistige Konzentration," etc., are frequently applied in such a way as unconsciously to make implicit these assumptions. They are often "nicht anderes als das noch immer seinen Spuk treibende Gespenst der Seele." The same authors who speak so glibly about these metaphysical centres are by no means unanimous as to the real existence of motor or sensory centres.

The author's personal contribution is divided into the three following sections:—

1. *Perception and Memory Images.*—External stimuli set up nerve processes, which are conveyed to appropriate parts of the cortex (stripe of Vicq d'Azyr, line of Gennari, etc.). Each stimulus finds its echo in a given cell or cell group, which is always the same for similar stimuli, but in addition it makes its influence felt in the whole layer of neighbouring cells. Pathological affections are hardly ever confined to such cell groups; however, the very existence of an enormous number of independent cells speaks strongly in support of this view. Wilbrand divides the perception of optical impressions into two parts, the perception field and the memory field. Jendrassik would not include the former, as no perception can occur without either the new formation or revival of memory images. In this process light and colour are

unessential accessories, form perception being the fundamental. Recognition of objects must, however, be independent of form perception if this, as Lissauer holds, is linked with activity of the oculo-motor nerves, for of course recognition is perfect with complete ophthalmoplegia. The dimensional analogy of photographs with memory images is incorrect, as is shown by the facts of stereoscopy; a tri-dimensional and "körperliche" formation of them must be postulated in the nerve-cells. It is thus more accurate to speak of memory figures than of memory pictures or images. The occurrence of corresponding images in both hemispheres is unthinkable, especially as each hemisphere is connected with both maculæ. Each image is represented in only one hemisphere, some in one hemisphere, some in the other. In some individuals the images may all be represented in the right hemisphere, in others all in the left. Macular vision is spared in a unilateral occipital lesion, but such a lesion may cause object-blindness of a limited nature. This contradicts the common teaching that only bilateral lesions can cause object-blindness. In Munk's experiments, often quoted in favour of the latter view, the occipital centres were preserved, and only their visual connections destroyed. Injury of these macular fibres—or of Vicq d'Azyr's layer, which, according to the author, is exclusively composed of these fibres—cannot, on the other hand, cause object-blindness unless it is bilateral, because the macular fibres of each side are connected with both occipital lobes.

The plan of the auditory system has many analogies with that of the visual. Certain differences are mainly in appearance only. For instance it would seem that multiple visual impressions are on the whole more often simultaneous than auditory ones. But on the one hand many auditory impressions may be instantaneously perceived, as when we recognise a person's voice or tone of an instrument, and on the other hand the visual perception of a large object is made up of a large series of rapidly succeeding impressions.

2. *Memory Association.*—An elementary psychological description of association is given. There is not the slightest ground for supposing the existence of an association centre; association is made possible by the existence of a complex layer containing impressions of memory images. The connections between these images do not consist of paths, but are brought about by means of a "tuning" of associated images, so that a stimulus evoking one, at the same time rings other images in harmony with this.

3. *The Arrangement of the Centres.*—The author holds that the Betz cells are purely motor, not sensori-motor, and contain no memory images of movement. In discussing the existence of a spinal writing centre he says that there is a priori no more reason

to deny the existence of such a centre any more than that of a special speech centre, using the term centre to indicate a group of cells that have been specially trained in each individual to perform certain functions. It is much easier thus to train sensory centres than motor ones; in other words criticism is easier than production.

Many interesting examples of highly systematised aphasias are given, such as loss of comprehension of all verbs except two, loss of capacity to write stenography only, etc. A case is quoted of an Italian child who stuttered on uttering Hungarian words beginning with ta, and no others; similar sounding words in Italian were pronounced well. Such examples prove to the author the existence of highly specialised centres, though he does not claim that these are necessarily sharply marked off from one another in the way that the motor centres are.

ERNEST JONES.

PATHOLOGY.

ON THE PATHOLOGY OF CHOKED DISC AND THE CHANGES

(195) **THAT FOLLOW TREPHINING. (Zur Pathologie des Stauungspapille und ihrer Veränderung nach der Trepanation.)** H. VON KRUDENER, *Graefes Archiv für Ophthalmologie*, Vol. lxv., p. 69.

THE knowledge of optic neuritis is being advanced at the present time by work done along two different lines. On the one hand the greater extension of intracranial surgery, and on the other hand researches on intracranial pressure, are contributing to put the genesis of the swelling of the optic disc in a clearer light. Krudener in this paper reviews the question from both those standpoints. He commences with a brief anatomical survey of the relations of the optic nerve and its sheath to the cerebral vascular and lymph systems on the one hand, and to the intraocular lymph system on the other. He thinks that too much stress has been laid by Seeseman on the anastomotic connections between the ophthalmic vein and the angular and facial veins and the pterygoid plexus, and he points out that there exist veins in the posterior part of the optic nerve which never get outside the dura mater in their course to the sinus cavernosus. Consequently obstruction in the sinus may lead directly to obstruction in the venous return from the posterior part of the nerve, and so indirectly to œdema in the anterior part, and in this way the vena centralis in its distal portion may be secondarily compressed. A part of his paper is devoted to very full clinical descriptions of a

few cases. From his examination of these he concludes that trephining produces a diminution of venous turgescence visible in about twenty-four hours. The diminution of the swelling begins on the second or third day. At the end of the second or third week there is always some swelling left. If the trephine opening closes up without removal of the original cause of the raised intradural tension, *e.g.* a tumour, the swelling of the papilla is soon as great as in the pre-operative stage. In one of his cases, in which there was an inflammatory neuritis consequent on a basal meningitis, trephining produced no result. The author concludes that trephining is distinctly called for in these cases, and he thinks that an even wider field will be found for it when it is possible to make a more accurate diagnosis between rise in intracranial pressure due to inflammatory and that due to non-inflammatory causes.

The results of lumbar puncture are negative. In no case was any effect, either subjective or objective, produced. The swelling remained unchanged, and the visual acuity, the fields, and the colour vision were in no way improved. None the less, Krudener thinks that in cases of optic neuritis, especially of a syphilitic nature, where all drug treatment has failed, lumbar puncture should be performed. Though no obvious ophthalmoscopic change is produced, the withdrawal of fluid will allow a greater blood flow to the diseased part, and consequently a more profound local action of the drugs.

He next describes four cases in which he made pathological examinations. In the first case, due to an echinococcus cyst in the left lateral ventricle, the nerve showed a marked œdema. The subarachnoid space was distended with fluid, and there were no inflammatory changes. The second case, a right cerebellar tumour, showed slight inflammatory changes, especially in the papilla and in the intracanalicular part of the nerve, but there was only a very small amount of small-celled infiltration. The third case, a left-sided brain tumour operated on with subsequent infection, died three weeks after the operation. There was wide-spread cerebritis and meningitis. The optic nerve and nerve sheath showed a rich round-celled infiltration. The fourth case (acute hydrocephalus) showed on examination similar inflammatory changes, an interstitial and parenchymatous neuritis with a leptomeningitis.

He concludes from the detailed examination of these cases that "inflammation alone is not the forerunner of choked disc, though in many cases of papillitis inflammatory appearances are found in the optic nerve. The *sine qua non* of the disc swelling is the raised intracranial pressure.

The concluding portion of his paper deals in a more general way with the etiology of choked disc and the theories as to its

causation. The simplest cause is where there is a general rise of the whole intracranial pressure, as is caused by the shutting off of the cranial lymph spaces from the vertebral, as in the case he instances by a tumour pressing back from the naso-pharynx; then the rise due to blockage of the aqueduct of Sylvius, *e.g.* by pressure of a growth of the pons or other neighbouring structures; then interference with venous return from the choroid plexuses. He shows that certain cases prove that there must be a further factor at work beyond those that are more purely mechanical, and some cases of hydrocephaly illustrate this where the initial lesion seems to lie more in the changes in the epithelium of the ependyma and meninges. In other cases the optic neuritis is not so much associated with a general rise of intracranial pressure as with some cause more local in its action, probably then acting through the orbital veins and vena centralis as explained in the earlier part of his paper. LESLIE PATON.

CLINICAL NEUROLOGY

CERTAIN ACUTE INFECTIVE OR TOXIC CONDITIONS OF THE

(196) **NERVOUS SYSTEM.** E. FARQUHAR BUZZARD, *Lancet*, March 16, 23, and 30, 1907.

THE Goulstonian lectures, by Dr E. F. Buzzard, deal chiefly with the pathology, and to a lesser degree with the symptomatology of acute anterior poliomyelitis, Landry's paralysis, acute toxic polyneuritis, and acute ascending—diffuse or disseminated myelitis. As a preliminary study, the first lecture is devoted to a study of the reaction of various elements of the nervous system to toxic agents.

Cell changes.—The toxic agent may produce alteration in the structure of the cell, either due to the "axonal reaction" or to primary affection of the cell protoplasm. Three types of appearances are recognised.

1. Early.—The cell hardly altered in shape, but the concave borders straight or slightly convex; the nucleus central or perhaps a little swollen; the chromatin granules showing either a perinuclear or a peripheral chormatolysis. 2. Moderate.—With a displaced nucleus and complete chromatolysis; in this stage recovery may take place. 3. Advanced.—Representing a fatal coagulative change.

Acute toxic changes in nerve fibres.—A Wallerian fatty degeneration of toxic origin is met with, associated with interference with the function of the fibre, which is, however, capable of restoration, and also the appearance of fine rounded drops of fat or material

stained by the Marchi method, common in the white matter of the cord. These drops lie within the myelin sheaths, but are seldom large enough to fill more than half of the transverse diameter of any particular fibre.

It has been observed in toxic states of all kinds. This change is not considered to be an evidence of loss of function.

Acute toxic changes in muscles.—Two varieties of appearances are met with: one a diffuse hyperplasia, characterised by the fact that the muscle fibre swells, so that its transverse diameter is increased and its longitudinal outline often rendered sinuous. The striation is generally lost. The second type is only recognised when the special fats stains, osmic, Scharlach red, or Sudan 3, are used.

These changes may be associated in one fibre. This alteration is especially common in diphtheria, in poisoning by the bacillus botulinus, and has been found by Buzzard in fatal ptomaine poisoning. It may be present without any alteration in the nerve fibre.

In normal conditions the adventitial or perivascular sheaths of vessels ramifying in the substance of the brain or cord may contain droplets of fat or granules of pigment. In toxic conditions the fat may be largely increased—it may be the result of chemical changes in elements of the vessel wall or abstracted from the nervous system.

Changes found in cases of toxæmia not associated with marked nervous phenomena.—The nervous system and muscles were examined in a number of cases of general toxæmia.

Very few definite changes were found in the cells of the cord or medulla, but the Betz cells of the cortex showed change in eight out of thirteen cases examined. The diffuse fatty change in the myelin was common in the series, but not the Wallerian type. The diaphragm and heart muscles more often showed fatty change than the skeletal muscles.

There was never any vascular cellular infiltration or neuroglial proliferation.

Paths by which bacteria or their toxins may reach the central nervous system and their means of dissemination within it.—The blood supply and lymphatics of the central nervous system are described. The important facts are: 1. Toxins in solution in the general circulation will be brought into closer relationship with the grey than white matter. 2. Solid particles forming emboli are rare in the cord, but occur in both the white and grey matter. 3. Organic and inorganic substances, when present in the lymph spaces of nerves, tend to be carried centralwards towards the spinal cord and meninges, and especially through the posterior roots. 4. That changes within the cord, the result of infection of

lymph channels, are patchy aud irregular, affecting the grey and white matter alike, with a special incidence on the posterior columns. 5. The morbid histological processes consist of toxic changes in ganglion cells, blood extravasations, small round-cell infiltration of tissue and perivascular sheaths, proliferating neuroglia, and parenchymatous degeneration of nerves.

Acute anterior poliomyelitis—morbid anatomy.—Buzzard takes, as the basis of his description, the case of a man, æt. 26, who died in thirty-six hours from the onset of paralysis, and two other cases in which death occurred within five days.

Summarising the changes he says: 1. The most widespread and constant features are, on the one hand, the congestion of blood-vessels and proliferation of the cellular elements of their adventitial sheaths; on the other hand, the absence of all evidence of ante-mortem thrombosis, or of changes in the internal coat. Most of cells appear to be derived from those already present in the walls of the vessels. 2. An intense proliferation or exudation of various cells concerning the origin of which there is difference of opinion, limited largely to those parts of the central nervous system most freely and generously supplied with blood—that is to say, in the neighbourhood of the rich capillary network of the grey substance of the cord and well-vascularised regions of the brain stem and brain.

These cells appear to be the following: 1. Large and small Deiter's cells of the neuroglia, a few of which present their normal characters, but the majority of which are altered. 2. Small rounded neuroglial nuclei and cells which resemble lymphocytes. 3. Round cell infiltration of the meninges extending in some cases to the intracranial meningeal cisterns.

Evidence of the infective nature of acute poliomyelitis is derived from the occurrence of epidemics, the seasonal occurrence of the disease, the identity of appearance in both the epidemic and sporadic cases.

Clinical.—Buzzard refers the cause of the pain seen in the beginning of the illness to the changes in the meninges, and points out that in some cases the change in the lateral columns of the cord is shown by the presence of the Babinski toe reflex.

Pathogenesis.—The interesting question as to the meaning of the vascular changes is discussed, and rejecting the "primary thrombosis" view, Buzzard considers that the lesion is an inflammatory one, that the toxin has an especial influence on the neuroglial tissue, and is more likely a hæmatogenous than a lymphagenous infection.

The bacteriology of the disease is at the present time unknown.

Acute Ascending Myelitis.—Buzzard relates a case: Man, æt. 44. Nine days before admission he noticed dull pain about the chest,

and the next day there was some weakness of the legs; this was followed by numbness of the legs, retention of urine, and increased weakness. On admission there was some fever; respirations 28 and diaphragmatic. There was weakness in raising the head from the bed, complete flaccid paralysis of the movements of wrists and fingers—paralysis of intercostals and abdominal muscles—legs flaccid and powerless, but no atrophy. Prick and touch were unperceived up to the level of the nipples, and only partially recognised as high as the fifth cervical root. No tenderness on squeezing the muscles—loss of the deep and superficial reflexes. Death from respiratory failure.

The results of the post-mortem showed congestion of the meninges and a diffluent condition of the cord, which on section showed brownish patches suggestive of extensive hæmorrhage.

A feebly Gram staining organism was present in the blood withdrawn during life and in the cerebro-spinal fluid and spinal cord.

Microscopically no changes were found in the sciatic nerve or in the anterior tibial muscle. In the cord the changes did not extend higher than the lower part of the pons. The most marked disease was found throughout the cord in the dorsal and lower cervical region. At every level foci of acute inflammation were present; in detail a section through the mid-cervical region showed the posterior median artery and its branches engorged with blood and surrounded in its adventitial lymph sheath by a cellular proliferation. In the white matter there was proliferation in the neuroglia. Myelin sheaths and axis-cylinders were swollen, anterior cornual cells were swollen and chromatolytic. The vessels showed no evidence of coagulation. Small hæmorrhages were present. Busch's method showed very few changes.

Buzzard considers that this disease is the result of a bacterial infection of the lymphatic system of the spinal cord. He considers that in the scattered irregularity of the lesions, the preference for the white matter, and especially for the posterior columns, and its spread in an upward direction along the centre of the cord and meninges, it resembles the morbid process produced experimentally by Homén when he injected organisms into the sciatic nerve or into the substance of the spinal cord.

Landry's Paralysis.—Buzzard gives the result of three post-mortem examinations. 1. No evidence of disease in the nervous system to the naked eye except general vascular engorgement. 2. Slight changes in the cells by Nissl method. 3. The fine scattered droplets in the peripheral nerves different from the true Wallerian degeneration. 4. No evidence of neuroglial or vessel change.

With the exception of changes in the ganglion cells, the morbid

appearances are those common to acute toxic changes of various kinds. Hence the poison acts chiefly on the ganglion cells, and these changes form the only obvious morbid anatomy of the disease. Bacteriological findings are discussed.

The clinical histories of these cases are given, corresponding to the generally recognised symptomatology.

Acute Toxic Polyneuritis.—Buzzard supposes that in these cases the poison is carried by the blood, and that whilst the cervical and lumbar enlargements suffer more than the dorsal region, the whole of each motor neurone is exposed to the poison circulating in the blood.

In Landry's paralysis, Buzzard, quoting Marinesco's experiments, suggests that bacteria or toxins present in the lymphatic system can produce toxic effects without inflammatory reaction.

Diagnosis.—The following points are given for the differential diagnosis between acute poliomyelitis and Landry's paralysis:—

1. Constitutional symptoms, fever, malaise, and anorexia more profound in poliomyelitis than in Landry's paralysis. Vomiting and gastric disturbance more frequent.
2. Pains about back, head, neck, and limbs more severe and urgent in poliomyelitis than in Landry's paralysis, in which there is more often discomfort or cramps.
3. Escape of a single or of a group of muscles suggests acute poliomyelitis, and within a few days rapid recovery occurs in some parts, with atrophy and electrical change in others. In Landry's the paralysis is evenly distributed, and recovery from it takes place slowly and evenly distributed without atrophy.

Between Landry's paralysis and acute toxic polyneuritis:—

1. Acute toxic neuritis may begin without any constitutional symptoms, which, though slight, are usually present in Landry's palsy.
2. In toxic neuritis, numbness, pins and needles, sharp pains, and tenderness of muscles, sometimes of the nerves, is the rule. Perhaps some anæsthesia of the glove or stocking type.
3. In toxic neuritis the limbs are more affected than the trunk, and the peripheral parts more than the proximal. The most marked incidence of palsy of the trunk muscles is on the diaphragm. Cranial nerves may be affected, especially the face and palate. Landry's palsy differs in its even and symmetrical distribution, in its progressive upward spread, in the involvement of the intercostal muscles before the diaphragm, and in affection of deglutition, phonation, and articulation.

4. Relapses and recurrences may occur in polyneuritis, but not in poliomyelitis or Landry's paralysis.

The final conclusion is that there are four distinct kinds of acute paralysis, which may be distinguished both clinically and anatomically. Acute poliomyelitis is an acute specific fever, in which the nervous system is attacked through the circulation, acute ascending myelitis, a spinal lymphangitis; Landry's paralysis, a species of spinal lymphatic intoxication and acute toxic polyneuritis, a toxæmia producing a parenchymatous degeneration of the lower motor neurones. W. B. WARRINGTON.

PROGRESSIVE HYPERTROPHIC INTERSTITIAL NEURITIS.

(197) **(Sur la névrite interstitielle hypertrophique et progressive de l'enfance; 2e observation suivie d'autopsie.)** DÉJÉRINE and ANDRÉ THOMAS, *Nouv. icon. de la Salpêtrière*, Nov.-Dec. 1906, p. 477.

THIRTEEN years ago Déjérine and Sottas described the cases of a brother and sister suffering from a disease which presented the following clinical symptoms: ataxia of the limbs, with muscular atrophy; marked sensory changes, with delay in the appreciation of stimuli; lightning pains, nystagmus, myosis, Argyll-Robertson pupil, and cyphoscoliosis; considerable hypertrophy and firmness of all such nerve trunks as could be felt; in a word, the symptoms were those of tabes, coupled with generalised amyotrophy, cyphoscoliosis, and hypertrophy of nerve trunks. The result of the pathological examination in the case of the sister, who died in 1892, was to reveal the existence of a hypertrophic interstitial neuritis affecting successively the peripheral nerves, nerve trunks, and anterior and posterior roots, with consecutive medullary lesions.

The brother died of phthisis in January 1901, and his clinical history and the result of the autopsy are recorded in detail in this paper. His mother had noticed curvature of his back when he was only five years old. At the age of fourteen it was remarked that his gait was a little irregular, that he was inaccurate in the use of his fingers, and that his speech was becoming affected. At the same time he began to suffer from severe lightning pains in his limbs, especially the legs. Apart from the fact that the titubation and oscillation in walking gradually increased, there was little change in his condition for some years. He contracted syphilis at the age of twenty-four.

When he entered Bicêtre in 1890, under Déjérine's care, the condition was briefly as follows: the feet presented a characteristic equino-varus on both sides, with extension of the proximal phalanges of the toes, and flexion of the distal ones. There was

considerable atrophy of the leg muscles, to a less extent of the thigh muscles. An extreme degree of ataxia was observed, whether the patient walked or stood still. The hands were typically simian, the forearms wasted, the muscles of the upper arm thin, but not particularly atrophic. In various muscle groups in arms and legs, fibrillation was present. Sensation in its various forms was greatly impaired in limbs and trunk, there being analgesia and anæsthesia, more intense distally than proximally. There was delay in the appreciation of all forms of cutaneous stimuli, and there was marked inaccuracy in the localisation of touch. The knee and triceps jerks were absent, and no response was obtained on stimulating the sole of the foot. There was more inco-ordination in the arms than in the legs. Both pupils were small, the right somewhat larger than the left, and they were typically Argyll-Robertson. On lateral excursion of the eyes, nystagmus was elicited. The sphincters were intact. The lightning pains were so severe that they sometimes brought the patient to an abrupt halt as he walked; sometimes he actually fell. There was marked diminution of faradic excitability in the affected muscles, but no reaction of degeneration.

Gradual increase in the symptoms took place during the next few years. In 1896, paralysis of the left vocal cord was observed. The left side of the face began to show indications of paresis, the lips began to pout, suggesting somewhat the myopathic facies. The optic discs remained normal, and the visual field was unrestricted. Phthisis pulmonalis was the cause of the patient's death in January 1901. Post-mortem it was found that the roots forming the cauda equina were at least twice as thick as the average, while the dorsal and cervical roots were also thicker than normal, though to a less extent than those of the cauda equina. The anterior were more hypertrophied than the posterior; in the case of the former, the hypertrophy decreased from the cord towards the theca; in the latter, the ganglionic end was thicker than the medullary end. All the spinal ganglia were enlarged; the sympathetic and cranial nerves were all larger than normal. The peripheral nerve trunks were very greatly augmented in volume, much more than the roots from which they came.

Microscopical examination of the affected muscles showed that, in the case of the thenar eminence, muscular striation was no longer recognisable; the nuclei were few, the fibres replaced by fatty vesicles or by fibrous or cellular tissue; all the blood-vessels had thickened coats. Where the muscles were less affected the atrophy was irregularly distributed; some fibres were atrophic, others were normal; most were broken up into pigmented masses, surrounded by nuclei, and stained unequally; between simple

atrophy and this myopathic condition all sorts of intermediate states were to be seen.

Longitudinal section of the muscles made it clear that the morbid process did not attack the fibres equally in their extent; it seemed as though groups of nuclei—often staining badly with hæmatoxylin—were breaking up the fibres into masses. In sections treated by Marchi's method, interstitial steatosis was not uniform or particularly marked anywhere.

Examination of the intramuscular nerve filaments revealed the extent to which they had been replaced by connective tissue, although even where they were most degenerated it was usually possible to distinguish one or two myelin sheaths and axis-cylinders still intact.

Sections of the nerve trunks were very instructive. The hypertrophy was almost entirely due to overgrowth of fibrous tissue in the nerve bundles and of the nerve sheaths; the epineurium was not very much changed. In the nerve bundles it was difficult to distinguish nerve fibrils, as so many had disappeared. Those left were surrounded by a sheath of imbricated connective tissue, inside which the myelin sheath might remain, still enclosing an axis-cylinder. The thickness of the connective tissue sheath was very variable; few nuclei were discoverable in it; it appeared to be highly organised, one might almost say cicatricial. In many of these hypertrophied sheaths no central myelin sheath or axis-cylinder persisted. The perineurium formed a dense sclerotic ring round each nerve bundle; sometimes these bundles were widely separated by connective tissue with a rather open meshwork. In many instances such myelin sheaths as remained were irregularly thickened, and by dissociation it was sometimes possible to remark hypertrophy of the sheath of Schwann. Various appearances noted by the authors suggested that the morbid process is of an ascending nature, as far at least as the anterior roots are concerned.

Most of the cells in the spinal ganglia were atrophic. Some of the blood-vessels in nerves and ganglia showed hyaline degeneration.

In the spinal cord there was typical degeneration of the posterior columns in their whole extent, with relative conservation of the cornu-commissural zones. The antero-lateral columns were normal. Only a few of the cells of the grey matter showed changes by Nissl's method. In brain, pons, and medulla no change was noted.

Of the existence of progressive hypertrophic interstitial neuritis as a clinical and morbid entity there can be no doubt. The histogenesis of the lesions is, however, somewhat obscure. The authors believe that the parenchymatous degeneration and

the interstitial hypertrophy develop simultaneously and independently, and that neither is secondary to the other.

The striking analogies between this condition and tabes cannot fail to be noticed; yet in the latter disease degeneration in the anterior roots, as marked as in the former, is infinitely rare; and of course hypertrophy of the roots is never seen. It has been maintained by Marinesco that hypertrophic interstitial neuritis and peroneal muscular atrophy (Charcot-Marie-Tooth) are identical, but this is obviously erroneous. Raymond considers the former a variety of the latter, but again this is quite untenable. On anatomical grounds alone no comparison is possible.

It should be mentioned that in none of the cases of hypertrophic interstitial neuritis quoted by the authors were sphincter difficulties noted, and this is a point of importance. No reference is made to the cases recorded by Brasch (this Review, 1904, p. 703). S. A. K. WILSON.

PERIPHERAL FACIAL DIPLEGIA AND PALATAL INVOLVE-
(198) **MENT.** GEORGE W. JACOBY, *Journ. of Nerv. and Ment. Dis.*, March 1907, p. 172.

FACIAL diplegia has been very fully described, but since observers differ as to the presence or absence of certain symptoms, the author here reports four cases examined by himself, more especially as these cases have a bearing on the views held as to the nerve supply of the palate. Only cases of peripheral origin are of importance in this connection.

The chief views held on the nerve supply of the palate are as follows:—

(1) It is derived from the facial through the Vidian and petrosal nerves (Davaine). (2) From the glosso-pharyngeus (Debron). (3) Motor from the facial, sensory from the glosso-pharyngeus. A lesion of the facial above the geniculate ganglion should therefore cause unilateral palatal paralysis of the same side. (4) The levator palati is supplied by the spinal accessory, in facial paralysis there is no defect of movement of the palate (Gowers). (5) "Bell's palsy with paralysis of the palate . . . I have never met with" (Hughlings Jackson). (6) Middle ear disease may lead to facial and palatal palsy by injury of the same nerve, and palatal palsy due to vagus affection differs from that due to facial affection (Maum). (7) Excitation of the facial causes no movement of the palate; excitation of the 10th and 11th pairs causes contraction of the palate. (8) Clinically lesions of the vago-accessory cause paralysis of the palate, but observers

differ as to the production of palatal paralysis through lesions of the facial.

With regard to the last point, the author suggests that cases examined early may show palatal involvement, while long-standing cases show none, the two halves of the palate being synergic, and therefore probably bilaterally represented in the cortex, and he claims to have shown elsewhere that even in peripheral palsies of such muscles some function may be re-established, when other muscles supplied by the affected nerve remain paretic. This, if accepted, explains the presence of palatal palsy in facial diplegia, and its rarity in unilateral facial palsy. Observers differ as to the frequency of palatal palsy in facial diplegia, but the author quotes four cases from his own experience, in two of which palatal palsy was complete and in one unilateral, while the fourth escaped. The value of all these cases, however, is much diminished by the fact that in all more than one nerve was involved; in one the sensation of the palate was affected, in another the sense of taste, and two were clear cases of multiple neuritis; they give little information, therefore, as to the innervation of the palate. D. W. CARMALT JONES.

HEMILINGUAL ATROPHY OF TRAUMATIC ORIGIN. SMITH (199) ELY JELLIFFE, *Journ. of Nerv. and Ment. Dis.*, March 1907, p. 194.

THE patient was thrown violently backward in a car accident. After the accident he held his head rigid, with the chin forward; active and passive movements were very painful. No evidence of nerve injury was detected. A month later his speech became thick, and the tongue became asymmetrical. The right half became broadened, moist, furrowed, and wasted, and showed fibrillary tremors. The whole tongue was protruded to the right, and there was bending of the raphé. The face was flushed and the right pupil dilated. There was no evidence of other nerve lesion. The diagnosis was injury to the hypoglossal nerve at its exit from the skull. D. W. CARMALT JONES.

MITRAL STENOSIS AND RECURRENT PARALYSIS. (Mitral-
(200) **stenose und Rekurrenslähmung.)** W. KOELLREUTER, *Monatsschr. f. Ohrenheilk.*, Bd. xli., H. 1, S. 1.

ORTNER in 1897 was the first to demonstrate cases (2) of recurrent paralysis caused by mitral stenosis. Both patients were examined after death. Hofbauer in 1905 described a similar case. Since

this date 9 cases have been published. Of these only 4 were examined post-mortem, and in none was the diagnosis confirmed. The author gives details of a case which was first considered to be the result of mitral stenosis, but further investigation negatived this view; it is hoped that a correct diagnosis will be arrived at after the patient joins the majority. W. G. PORTER.

DIAGNOSIS AND LOCALISATION OF CEREBRAL TUMOURS.
(201) CHARLES E. BEEVOR, *Lancet*, Feb. 9, 23; March 16, 1907.

THE problems of cerebral localisation have been studied by three different methods—experimental, histological, and clinical. Experimental research has led the way, and, following in its train, histological and clinical studies have advanced far towards the solution of many of the problems. In his Lettsonian Lectures, Dr Beevor reviewed shortly the facts which have been ascertained by experimental and histological investigators, and pointed out their influence in placing clinical study in a more scientific, and therefore more useful, position. His lectures were chiefly concerned with the clinical and practical aspect of his subject. He dealt with his subject upon broad lines, taking each region of the brain in turn and illustrating the symptomatology and diagnostic points characteristic of tumour of each area by giving an account of groups of typical cases and summarising the clinical findings. Clinical study can never hope to attain the exact results which are possible in experimental work, but it has two advantages. Firstly, it studies the conditions as presented to the physician—the effect of lesions produced by disease, and as such often complicated secondly, the examination of patients permits of extensive inquiry into the psychical and sensory phenomena which experimental research on animals can never elucidate.

Before entering into the consideration of the localising signs of cerebral tumours, Dr Beevor described the various general symptoms—headache, vomiting, and optic neuritis—and discussed their value as diagnostic signs of intracranial growth as against other conditions, and explained the importance of the association of local cerebral signs, paralytic or irritative. It is impossible in a review to quote any of the excellent cases by means of which Dr Beevor illustrated the effects of tumour in the various regions; but it is well worth while to summarise briefly the characteristic diagnostic points to which he drew attention.

Tumours of the Motor Region.—(1) Ascending frontal convolution, also tumours involving both the ascending frontal and the ascending parietal convolution. Besides the general symptoms of ntracranial neoplasm, tumours of this region give rise to (*a*)

irritative and (*b*) paralytic symptoms. The irritative symptoms are fits of Jacksonian type which are sometimes hemiplegic, sometimes becoming general, commencing with movements on the side opposite to the lesion. If the tumour involves the ascending parietal convolution, then a local sensory aura may be present and corresponding sensory affection. The paralytic symptoms are paresis or paralysis on the opposite side local (face, arm, leg), or hemiplegia with or without hemianæsthesia, the latter depending upon the affection of the ascending parietal convolution. As a rule paralysis is observed after a fit, especially in the part most affected, and a fit may commence with movement of a paralysed limb. If the tumour be situated deeply, then there is less tendency towards local irritative symptoms, the characteristic feature being a slow progressive spastic hemiplegia.

Tumours of the Frontal Region.—These were considered under the following grouping: 1. Tumours outside the brain. 2. Tumours growing in the cortex on the outer surface of the brain, (*a*) situated anteriorly, (*b*) situated posteriorly. In addition to the general symptoms, mental symptoms are prominent and characteristic, dulness, loss of memory, inattention, incontinence of urine, etc. The focal symptoms are faints simulating petit mal attacks when the tumour is anterior in situation; local Jacksonian attacks, commencing with turning of the head and eyes to the opposite side, or with clonic movements of the face, arm, leg, etc., on the opposite side; in left-sided cases, motor aphasia attacks are met with. General epileptic fits are also not uncommon. The localising signs are: (1) loss of smell on the same side, if the tumour be situated on the orbital surface of the lobe; (2) paresis or paralysis of the conjugate movements of the head and eyes to the opposite side, or weakness of the face, etc., on the opposite side; (3) a fine vibratory tremor in the limbs on the same side as the tumour; (4) increase of the deep reflexes, loss or diminution of the superficial reflexes on the opposite side, with sometimes an extensor plantar response. 3. Tumours growing in the substance of the frontal lobe do not cause loss of smell, but may later give rise to paretic symptoms on the opposite side and corresponding changes in the reflexes. Dr Beevor holds strongly that mental symptoms are not more marked in cases of tumour of the left frontal region than in cases of tumour of the right frontal region.

Tumours of the Temporal Lobe.—In cases where the tumour is situated in the temporal lobe, the localising signs vary according to the position of the growth. The "dreamy state" and sensations of smell and taste point to involvement of the uncinate and anterior part of the lobe, while loss of hearing in the opposite ear points to involvement of the superior temporal convolution. Besides these signs, slight motor weakness of the face or limbs, with increase in

deep reflexes and diminution of the superficial and a plantar extensor response on the opposite side, due to secondary pressure of the growth on the internal capsule, often give help in the localisation. Word-deafness occurs in tumours of the superior temporal region situated on the left side.

Tumours of the Supra-marginal and the Angular Gyri.—The general symptoms are often late in appearing. The local signs are fits, petit mal, Jacksonian attacks with sensory auræ affecting the opposite side of the lobes, or general convulsions. Hemianæsthesia and analgesia of varying degrees is present, and often hemianopia of the opposite field, when the growth extends to involve the optic radiation. When the tumour is on the left side, word-blindness is produced.

Pituitary Tumours.—These give rise to the general symptoms of intracranial neoplasm, and also cause impairment of memory. The most important localising sign arises from the pressure of the growth upon the optic chiasma, which is characterised clinically by the occurrence of bi-temporal hemianopia. Acromegaly may result in some cases, but this is by no means common.

Tumours of the Optic Thalamus, if situated in the posterior part, give rise to involuntary movements and ataxia of the opposite limbs, voluntary movements not being much affected, unless the internal capsule is pressed upon. The deep reflexes of the opposite side are increased, the superficial reflexes are normal or diminished, and the plantar response is flexor in type provided the internal capsule is not involved. Sensory symptoms are prominent, subjective sensations of pain, heat, and cold are frequently met with, and hemianæsthesia, loss of muscular sense, and stereognosis are found on the opposite side. Hemianopia is only met with if the optic radiations are pressed upon.

Tumours of the Sub-thalamic Region, involving the red nucleus, give rise to ataxia and a more or less rhythmical tremor of the opposite limbs.

Tumours of the Corpora Quadrigemina are localised by the affection of the ocular movements, especially depending for their movements on the third nerve. Ptosis, loss of upward and downward movements, convergence with sluggish or lost reaction of the pupils to light. Staggering to the side opposite to the lesion is also met with in tumours of the corp. quad. on one side.

Tumours of the Cerebellum.—These were considered in the following groups: (1) extra-cerebellar tumours; (2) intra-cerebellar tumours, (*a*) of the lateral lobe, (*b*) of the vermiform process.

(1) Extra-cerebellar tumours. Optic neuritis, later in appearance. Vertigo, objects move from the side of the lesion, patient has feeling of rotation of self towards the side of the lesion. Deafness and facial paralysis on side of lesion. Affection of sixth

and fifth nerves on side of lesion. Lateral horizontal nystagmus to side of lesion. Paresis (cerebellar) and ataxia on same side. Paresis (pontine) and ataxia on opposite side due to pressure upon the pons, increase in the deep reflexes, and often extensor plantar response on side opposite lesion.

(2) Intra-cerebellar tumours. (*a*) Of lateral lobe. Weakness of conjugate movements of eyes and horizontal nystagmus most marked to side of lesion. Paresis, ataxia, loss of tone on same side as lesion. Never complete deafness; subjective and objective movements of vertigo are both in same direction,—away from the side of the lesion. Plantar reflex is never extensor. (*b*) Of the vermis, give rise to reeling gait, tendency to fall backwards, and nystagmus. T. GRAINGER STEWART.

BRAIN TUMOUR SYMPTOM-COMPLEX, WITH TERMINATION
(202) **IN RECOVERY.** HERMAN H. HOPPE, *Journ. Nerv. and Ment. Disease*, Feb. 1907.

THIS is a report of three cases in which there was the symptom-complex of brain tumour. In one an autopsy revealed no morbid condition; in the other two cases recovery has taken place. The author refers to the literature on this subject, and discusses the various theories which have been advanced to explain the condition. T. GRAINGER STEWART.

CEREBRAL EMBOLISM IN DIPHTHERIA. (Hirnembolie im Ver-
(203) **laufe der postdiphtherischen Herzschwäche.)** ESCHERICH, *Wien. med. Woch.*, March 2, 1907, p. 478.

ESCHERICH records two cases of cerebral embolism in diphtheria, associated with the characteristic cardiac involvement. The first case was that of a boy aged 2 years, who was admitted to hospital on the sixth day of a severe attack of faucial diphtheria with broncho-pneumonia. Right hemiplegia developed the same day. The next day generalised convulsions occurred. On the eighth day the thighs became cold, livid, and devoid of sensation. No pulse could be felt in the popliteal and dorsalis pedis arteries. Death took place the same day. At the autopsy a thrombus was found adherent to the ventricular surface of the mitral valve. There was embolism of the left middle cerebral artery, with recent softening of the left cerebral hemisphere. Emboli were also found in the left common iliac artery at its bifurcation and in the right femoral artery. Small recent infarcts were present in the right kidney and in the liver.

The second case was that of a boy, aged 9 years, who also had a severe attack of diphtheria followed by paralysis of the palate and pharynx, loss of accommodation and squint. Three weeks after the onset of the disease he had an apoplectic attack, with loss of consciousness and general convulsions. At first all the four limbs were paralysed. In the course of the next month, as the other palsies disappeared, the power of movement in the left limb returned, but the muscles of the face and limbs on the right side still showed a spastic paralysis. In the date of onset of the hemiplegia, in its association with the characteristic palsies of diphtheria, and in its incomplete recovery, this case offers a striking resemblance to that reported by the abstracter (*Review of Neurology*, 1905, p. 722). J. D. ROLLESTON.

HÆMORRHAGE FROM THE SINUSES OF THE DURA MATER.
(204) J. HOGARTH PRINGLE, *Scot. Med. and Surg. Journ.*, March 1907.

SIX cases of this condition are recorded, each the result of severe injury. In two, rupture of the sinus was caused by distortion of the skull at the moment of the infliction of the violence; in three there was direct injury by penetrating fragments of bone; and in one the injury was the result of a pacchionian body being torn out of the sinus. None of the cases presented a localised extra-dural clot. In four cases the superior longitudinal sinus was injured, and in two the lateral sinus. Four of the patients died, one in consequence of sinus hæmorrhage alone. In the writer's experience there had occurred 16 cases of ruptured meningeal artery due to head injury, as compared with 6 of sinus injury.

Injury to a sinus may be due to penetration by a foreign body or a fragment of bone, or may be produced indirectly by distortion of the skull from violence. Fracture of the skull is not a necessary accompaniment. Rupture of the longitudinal sinus may occur during birth as the result of compression by a narrow pelvis. One case is on record in which spontaneous rupture of the lateral sinus caused death. The effused blood may escape through a fracture and form a hæmatoma under the scalp, or may find a free exit altogether if the scalp be wounded as well. A large extra-dural accumulation is rare, the pressure in the sinuses not being great enough to force out much blood. If the sinus be injured on its cerebral aspect only, an extensive subdural hæmorrhage may form rapidly and lead to general or local compression of the brain. Diagnosis of sinus hæmorrhage is very difficult unless there is a direct communicating wound. In all cases of fracture involving the region of a sinus, the possibility of injury to it should be borne in mind, as a penetrating fragment may plug the opening, and

hæmorrhage may not occur till this is removed. In the treatment the aim is to arrest hæmorrhage, to relieve brain pressure when it is present, and to preserve asepsis. HENRY DUNBAR.

TWO CASES OF CEREBRAL SYPHILIS SIMULATING TYPHOID FEVER. (205) J. D. ROLLESTON, *Medical Press and Circular*, March 20, 1907, p. 307.

THE mental symptoms of syphilis, especially if associated with pyrexia, may occasionally lead to the mistaken diagnosis of typhoid fever. Such cases are rare, as is shown by the fact that out of 3076 cases in the last 10 years sent to the Metropolitan Asylums Board Hospitals, wrongly certified to be suffering from typhoid fever, only 10 were diagnosed after admission as syphilis. Syphilologists also testify to the rarity of the condition. The first of the two cases recorded by the writer was that of a man, aged 35, sent to hospital certified to be suffering from typhoid fever. A healing chancre was present on the penis and a polymorphic eruption on the trunk. The liver was enlarged. The temperature was of a low grade and of an intermittent type. Bilateral ptosis, most marked on the right side, was present. Violent delirium persisted during his six days' stay in hospital. Large doses of mercury and iodide had little effect. Death took place in an infirmary three weeks later. Business worries seem to have been the determining cause of the cerebral syphilis. The second case was that of a girl, aged 19, also sent to hospital as a case of typhoid. There was no violent delirium in this case, but the patient presented the dazed appearance and thickly coated and tremulous tongue often met with in typhoid fever. The temperature was high. The eruption, however, did not resemble the rose spots of typhoid but the roseola of syphilis. Mucous tubercles and inguinal adenopathy were also present. Small doses of mercury were given, and the temperature, which for the first three days was 103°, reached normal on the fourth day, and became finally settled on the sixth. With the fall of the temperature the mental obnubilation cleared away. From a diagnostic point of view it is of interest that in both these cases the abdominal reflex was active (vide *Review of Neurology*, 1906, p. 582). AUTHOR'S ABSTRACT.

CEREBRO-SPINAL MENINGITIS. (206) WILLIAM ELDER and NENA IEVERS, *Scot. Med. and Surg. Journ.*, March 1907.

FIVE cases are recorded, four of them in children under 9 years, and one in a man aged 37 years. In all the diplococcus was demonstrated in the cerebro-spinal fluid, and in four a typical

purulent cerebro-spinal meningitis was found on post-mortem examination. The symptoms may be described as being a very acute and marked exaggeration of those present in the more common tubercular meningitis. Cramps and twitchings are always present; head retraction and convulsions may occur, but not always. The temperature rises to 101° or 102°, or even to 104°; the pulse is rapid (140-180) and usually irregular; and the respirations do not usually reach beyond 40, and are often jerky in character. Purpuric spots may or may not be present; they usually appear if the patient survives long enough. Lumbar puncture is the only sure means of diagnosis. Of the five cases, one recovered and one made a temporary recovery. Both of these patients received injections of De Rienzi's anti-pneumococcic serum. One of the fatal cases received an injection when almost moribund; in the others it was not administered. In the two cases a distinct improvement was noted after each injection. This treatment was instituted in the first case under the impression that the disease was of pneumococcal origin. The writer thinks that the meningococcus may be closely allied to the pneumococcus, and in support of this opinion refers to the variations shown by the meningococcus in culture, particularly the change from Gram negative to Gram positive. HENRY J. DUNBAR.

NOTE ON TWO FATAL CASES OF ACUTE MENINGOCOCCAL
(207) **CEREBRO-SPINAL MENINGITIS IN YOUNG CHILDREN, WITH PATHOLOGICAL REPORT ON ONE OF THEM.** JOHN THOMSON and STUART M'DONALD, *Scot. Med. and Surg. Journ.*, March 1907.

THE first patient was a boy, aged 3½ years, who had been taken suddenly ill two days before admission to hospital. The symptoms had been vomiting, feverishness, and thirst, difficulty in breathing, slight cough, restlessness, headache, and finally stupor. After admission it was noted that he lay on his side with his limbs flexed, quite unconscious of his surroundings. When disturbed in any way he screamed loudly. There was no rash. The pulse was 152, respirations 52, and temperature 102°. There was retraction of the abdominal wall, and the neck and back were rigid. The knee-jerks were absent, and Kernig's sign was well marked. Turbid fluid under considerable tension was obtained by lumbar puncture, and was found to contain many cells, mostly polymorphs, and numerous diplococci, both extra- and intra-cellular. The diplococci were typical in appearance, and were Gram negative. The child died on the day of admission, but no post-mortem was allowed. The second case was that of a girl aged $2\frac{2}{12}$ years, who had been

ill for 2½ days before admission. She exhibited more marked head retraction than the first case, and opisthotonos was present. The knee-jerks were normal, and Kernig's sign was absent. There was no rash. There was great drowsiness, but the patient seemed conscious. The temperature was not observed to rise above 99·2°. While in hospital, lumbar puncture was performed twice, and appeared to give considerable relief the first time. The fluid obtained was turbid, and contained the meningococci. There was a leucocytosis of 19,000, with 70 per cent. of polymorphs. The patient died on the day following admission. Post-mortem examination revealed a wide-spread leptomeningitis, with yellow exudate over the vertex, sides, and base of the brain, and the spinal cord, especially over the cervical and lumbar enlargements. In this case (as in another one observed later) the organism was Gram negative in films from the tissues, but became Gram positive in culture. The possibility of mixed growth could be excluded. The organism found in this disease is identical with that occurring in cases of posterior basal meningitis, but in a more virulent state.

HENRY DUNBAR.

NEURALGIC AFFECTIONS OF THE GENICULATE GANGLION
(208) **AND ITS DIVISIONS (OTALGIA). A PRELIMINAY REPORT.** J. RAMSAY HUNT, *Journ. of Nerv. and Ment. Dis.*, Feb. 1907.

THIS author gives a short note upon the connections of the facial nerve and ganglion—(1) with the interior of the auricle and external auditory canal; (2) with the tympanic plexus; (3) with the second and third divisions of the fifth nerve; (4) reflexly with the glosso-pharyngeal; (5) reflexly with the vagus, showing that there is an anatomical basis for the numerous and various forms of reflex otalgia. But in addition to these reflex neuralgias the author holds that there is a primary otalgia due to a specific affection of the geniculate ganglion, and not, as has previously been maintained, to an affection of either the fifth or the second and third cervical nerves.

T. GRAINGER STEWART.

ON HERPETIC INFLAMMATIONS OF THE GENICULATE GAN-
(209) **GLION. A NEW SYNDROME AND ITS COMPLICATIONS.** J. RAMSAY HUNT, *Journ. of Nerv. and Ment. Dis.*, Feb. 1907.

UNDER the general heading of herpetic inflammations of the geniculate ganglion, the author has brought together three separate groups of cases. He has collected 56 cases from the literature on

the subject, and adds 4 cases from his personal observation. The three groups which he unites to form a definite clinical entity are:—

1. Herpes zoster auricularis.
2. Herpes zoster in any of the zoster zones of the cephalic extremity (herpes auricularis, herpes facialis, and herpes occipito-collaris), with facial palsy.
3. Herpes zoster of the cephalic extremity, with facial palsy and auditory symptoms (tinnitus, deafness, vertigo, vomiting, nystagmus, and disturbances of equilibrium).

His argument is based upon the fact that the facial nerve is a mixed nerve, and that its sensory ganglion is the geniculate ganglion. The motor root of the geniculate is the facial nerve proper, and its sensory root is the nerve of Wrisberg. Below the ganglion, the peripheral divisions are: the facial nerve proper, the great and lesser superficial petrosal nerves, the external petrosal, and the chorda tympani. The only neural connections existing between the geniculate ganglion and the cutaneous area (described as its zoster zone) are: (1) the auriculo-temporal branch of the fifth nerve through the medium of the small superficial petrosal nerve and otic ganglion; and (2) through the facial nerve proper. In the author's opinion the facial root is the probable course taken by the afferent fibres from the auricle. The Gasserian, geniculate, and the ganglia of the second and third cervical nerves form a ganglionic series in every way similar to the posterior ganglia of the spinal roots. They are of the same type, and as such liable to true herpes zoster. Further, as in herpes zoster of the spinal ganglia, while one may be chiefly affected, the neighbouring ganglia may be involved to a lesser extent, so in the case of these higher ganglia. The zoster zone of the geniculate ganglion lies between that of the gasserian and the second and third cervical ganglia, and is situated within the auricle and external auditory canal. In all Krause's cases, and in those of Frazier and Spiller, in which the Gasserian ganglion was extirpated, no loss of sensation was made out within the auricle or external auditory canal. In Cushing's cases, where sensory loss was observed, the author thinks that during the tearing out of the ganglion the connections between the fifth nerve, Weckel's ganglion, the otic ganglion, and the Gasserian ganglion may have been interfered with. Cushing's observations of the loss of sensation caused by extirpation of the second and third cervical ganglia show that there is no loss of sensation within the auricle or external auditory canal. The facial paralysis and the auditory symptoms are to be explained by either a direct extension of the pathological process to the nerves themselves or to their involvement indirectly by pressure, all being in a common sheath and lying in a bony canal.

The pathology of the affection is the specific hæmorrhagic inflammation of the ganglion as found in zona elsewhere.

The clinical types of the disease are described and two cases are reported. A pathological examination of one of these, 87 days after onset of the disease, showed with Marchi: (1) Above the ganglion, degeneration of the nerve of Wrisberg alone, passing along the course of the internal root of the auditory nerve to the fascialis solitarius and the central grey column of the medulla, the facial nerve being healthy. (2) Below the ganglion, degeneration of the facial nerve peripherally. Unfortunately the geniculate ganglion was not obtained for examination in this case. The cells of the facial nucleus, stained by Nissl, showed early degenerative changes. T. GRAINGER STEWART.

FAINTS AND FAINTING. Sir WM. GOWERS, *Lancet*, March 2, 1907 (210) p. 565.

THE author calls attention to our ignorance of the intimate mechanism of this process. It certainly is not a mere question of cutting off nutrition from the brain substance. Except in fatal cases, cardiac syncope probably never causes absolutely sudden loss of consciousness. In contrasting the condition with minor epilepsy, the following points are to be noted. On recovery from syncope there is from the first correct appreciation of surroundings, but some slight physical prostration; after petit mal there is no physical prostration, but often some mental confusion, though this is by no means invariable. Pallor never occurs at the onset of petit mal, and often not throughout the attack. Minor epilepsy attacks are often diagnosed as syncopal, owing to the tendency of the mind to diagnose the less grave condition. Epigastric auræ are often incorrectly interpreted as cardiac manifestations. The nature of an exciting cause may yield clues; thus, for instance, change to an upright posture often induces syncope.

The author regards it as certain that repeated syncopal attacks may pass into true epilepsy, and quotes three illustrative cases of this. ERNEST JONES.

PSYCHASTHENIC ATTACKS SIMULATING EPILEPSY. WILLIAM (211) G. SPILLER (of Philadelphia), *Journ. of Abn. Psych.*, February 1907.

THE author draws attention to psychasthenic attacks which have frequently been looked upon as manifestations of either epilepsy or hysteria, and which he thinks should not be classified with either. He refers to contributions which have been made to the

subject by Westphal (1872), Oppenheim (1905), and Janet (1906). Westphal referred to such attacks as being frequent accompaniments of psychopathic and neurotic states. Oppenheim considers these individuals to be neuropathic or psychopathic from birth; under the stress of overwork, excesses, sleeplessness, and anxiety these attacks develop. He excludes hysteria, because all hysterical stigmata are wanting. They resemble epileptic attacks much more, but may be differentiated by the following symptoms: The absence of such attacks in childhood; psychopathic traits such as tics, anxiety, phobias, which antedate the convulsions; a special cause to precipitate the attack; as a rule few or but one attack during the entire life; profound unconsciousness without convulsive movements or slight twitching limited to a few muscles; intelligence and memory unimpaired, although attacks may have been numerous. Janet discusses the features in common between psychasthenia and epilepsy.

Putnam and Waterman (1905) have referred to the possibility of certain forms of epilepsy and hysteria being more closely related than has been hitherto assumed; in this they have the support of several other writers.

Spiller gives in some detail the histories of two of his own cases which to his mind are neither epilepsy nor hysteria. In the first case he noted a neurotic family history; an aura, associated with bad taste in the mouth and ocular disturbances, which always preceded by one day the visual hallucination; the occurrence of the hallucination in one place only at first, but later in other places; the resemblance of the face seen to that of a boy who taught the patient masturbation; the frequency of attacks proportionate to the frequency of masturbation; drowsiness, fulness of the head and palpitation following an attack; absence of all convulsions. The treatment, which consisted in assuring the patient that the attacks were not serious, and in arousing his resistance to them, was to a degree successful.

The second case resembled epilepsy, but could not be so considered if carefully studied. The patient came from neurotic stock, and broke down under overstudy; numerous (25) convulsive attacks were observed—weak spells, staring, mind-wandering without unconsciousness. In speaking of these the patient declared that he was never unconscious, that the rigidity was voluntary, it was a temporary relief to faint, he made no mental effort to avoid staring at a spot on the wall when he felt like doing so, it was not real headache that he experienced, only consciousness that he had a head; if he had known that falling would have really injured him he never would have fallen, etc. The patient presented no hysterical stigmata nor signs of organic disease. The treatment was psychotherapy. C. H. HOLMES.

A STUDY OF THE SENSORY SYMPTOMS OF A CASE OF (212) **POTT'S DISEASE OF THE CERVICAL SPINE.** FRANK R. FRY, *Journ. of Nerv. and Ment. Dis.*, March 1907, p. 185.

THE author gives a *résumé* of Dr Head's work on protopathic and epicritic sensation; he employed the same methods in the investigation of this case.

The patient's first symptoms were pain in the occiput, and paræsthesiæ in the left arm. Movements of the head were limited, there was pain on pressure over the second and third cervical spines, there were no objective sensory symptoms, the deep reflexes of the left arm and both legs were increased. A year later there were paræsthesiæ in both arms and over an area on the right thigh, there was some difficulty in performing fine movements. On the upper border of the left trapezius was a small area which was hyperæsthetic to pin pricks and to heat and cold, but partly insensitive to light touch, warmth, and cold. There was a similar area on the deltoid. There were paræsthesiæ in the hands, but no objective changes. Later there were muscular jerkings in the right arm and leg, the gait became spastic, and a slight Babinski reaction appeared on the right. Spacing sense diminished at the tips of the fingers, the right arm and leg grew weaker, and over the paræsthetic area on this thigh there was slight loss of light touch sensibility and of temperature sense, and the toes were subjectively and objectively cooler.

Sensation gradually diminished over the upper and lower extremities, and the paralysis and sensory loss increased; small variable areas of protopathic and epicritic disturbance were found on the arms.

Sphincter disturbance ultimately supervened. At this time finer sensation was lost in the digits, position sense was lost, but pain, heat and cold, and deep pressure were only obtunded. It is remarked that the changes were most marked distally, and decreased on approaching the trunk.

The X-rays showed some disturbance of the cervical vertebræ, and a diagnosis of spinal caries was made.

The paper shows a very careful study of sensation, but no conclusions are drawn as to its value in diagnosis or otherwise.

D. W. CARMALT JONES.

ÆTIOLOGY AND SPECIFIC TREATMENT OF GRAVES' DISEASE, BASED ON PRACTICAL EXPERIENCE OF MOEBIUS' ANTITHYREOIDIN. (Zur Ätiologie und spezifischen Therapie des Morbus Basedowii nach praktischen Versuchen mit Antithyreoidin-Moebius.) RATTNER, *Neurolog. Centralbl.*, March 1, Jahrg. xxvi., S. 201.

(213)

THE author first expounds the two rival hypotheses of this disease now current, the primary medullary changes and the primary perverted thyroid secretion. He holds the latter as the more probable, and thinks the medullary changes may be secondary phenomena. The thyroid change is not a simple hypersecretion, as the disease cannot be produced by thyroid medication, and often it is actually improved by this.

He has treated a series of cases, four of which are described in detail, with Moebius' serum. The results observed were as follows: The three cardinal symptoms were sometimes slightly improved, but never for long. The diarrhœa, sweating, tremor, and sleeplessness were improved, but most striking was the change in the psychical symptoms. These in every case diminished, an experience identical with that of most observers.

The author very critically reviews his results and those claimed by others. He had previously been greatly impressed by a severe case in which all the symptoms, even the physical, showed a most striking improvement after a successful issue to a difficult love affair. Such variations in the disease are not rare. He queries how far the effect of Moebius' treatment on the psychical symptoms is due to suggestion. Certainly the treatment is in no sense specific. No cure has ever been recorded, even in cases treated for years.

ERNEST JONES.

A STUDY OF THE PARATHYROID GLANDULES IN PARALYSIS AGITANS. THOMPSON, *The Journal of Medical Research*, Dec. 1906.

(214)

THE author, after reviewing shortly our present knowledge of the history, structure, and function of the parathyroid glands, epitomises the results in the words of Jeandelize, "Insufficiency of the thyroid causes chronic troubles (nutrition), while insufficiency of parathyroids causes acute troubles (convulsive troubles)."

The present work was undertaken in order to see definitely whether there was morphologic reason for the hypothesis of Lundborg and Berkeley that paralysis agitans is a chronic, progressive, hypoparathyroidismus.

The results are based upon examination of the glands from 9 cases of paralysis agitans, with controls from 40 routine post-mortem examinations.

In 33 of the 40 control cases 4 glands were found, in 4 cases only 3, and in 1 case (a woman, æt. 70 years, who died from cerebral hæmorrhage) no glands at all could be discovered.

As regards the size, shape, colour, position, and histology of these glands the author has nothing new to mention.

After examination of the cases of paralysis agitans the author sums up: "Little is to be said save that the parathyroid glandules, in individuals dying with this disease, present no changes either in number, size, position, or histologic structure that would serve to distinguish them from the parathyroid glandules in individuals dying from other diseases. . . ."

"In conclusion, then, it must be stated that there is no morphologic ground for the assumption that the parathyroid glandules are insufficient in paralysis agitans."

A. DINGWALL FORDYCE.

MIGRAINE AND HEMIANOPSIA. JOHN JENKS THOMAS, *Journ.* (215) *of Nerv. and Ment. Dis.*, March 1907, p. 153.

UNDER this heading the question is discussed whether attacks of migraine, in the absence of vascular disease, can produce permanent changes in nervous structures. No definition or detailed description of migraine is given.

Moebius distinguishes idiopathic and symptomatic forms of migraine, remarks the frequent coincidence of migraine and vascular degeneration especially in the aged, and concludes that migraine may be the cause of organic brain disease.

Idiopathic migraine is often hereditary and begins in youth, and is essentially intermittent in character. The headache is typical, and to be distinguished from that of neurasthenia, syphilis, and organic brain disease. Symptomatic migraine may occur in tabes, G.P.I., or cerebral tumour. Recurrent oculo-motor palsy is a distinct though allied condition.

Hemianopsia is a common aura in migraine; transitory aphasia, hemiparesis, and hemianæsthesia also occur, and in rare cases become permanent, perhaps as a result of vascular spasm, but analysis of the reported cases suggests that in many of them a symptomatic migraine accompanied organic disease. Four cases of Galezowski's are quoted, two of permanent hemianopsia from arterial thrombosis or embolism in young subjects, occurring in migraine attacks.

Other cases of permanent hemianopsia or aphasia are quoted from literature, chiefly occurring in elderly patients.

Three cases of the author's own are described, which occurred in women between the ages of twenty-seven and thirty, all of whom developed permanent hemianopsia during typical migraine attacks.

The author concludes that while in the majority of the cases described vascular or cerebral disease existed, and the migraine was symptomatic, there is reason to believe that, as a result of idiopathic migraine, vascular lesions may occur in young persons with healthy vessels. D. W. CARMALT JONES.

APHASIA AND DEMENTIA WITH PROBABLE GENERAL
(216) **PARALYSIS. (Aphasie et démence avec paralysie générale probable.)** BOULENGER, *Journ. de Neurol.*, March 5, T. xii., p. 86.

A FEMALE patient, aged 29, was admitted in a state of mental confusion and excitation, and having a history of epileptiform attacks, mutism, and facial paralysis. In the next few days were observed aniscoria, exaggerated reflexes, and severe hystero-epileptiform attacks. There was a typical motor aphasia made up of dysarthria and paraphasia. This gradually improved, but a few months later word-deafness and increasing dementia appeared.

The diagnosis between gumma, cerebral syphilis, and general paralysis is discussed. ERNEST JONES.

THE LOSS OF THE KNEE-JERK IN LOBAR PNEUMONIA IN
(217) **CHILDREN. (Ueber das Westphal'sche Phänomen bei krüpposer Pneumonie der Kinder.)** KEPHALLINÓS, *Münch. med. Woch.*, July 24, 1906, p. 1460.

PFAUNDLER in 1902 (*Münch. med. Woch.*, No. 29) examined 200 children, between the ages of 1 and 13, suffering from lobar pneumonia, and found the knee-jerks affected in 55 (27·5 per cent.). Kephallinós has analysed 65 more cases of lobar pneumonia from Pfaundler's clinic in children between the ages of 4 and 14. The knee-jerks were affected in nearly 50 per cent., being entirely absent in 27 cases (41·5 per cent.), much diminished in 3, and absent on the right side only in 2. The absence of knee-jerks was eight times more common than the presence of herpes labialis. In several cases the knee-jerks were found to be absent before the appearance of any signs in the lungs. Out of 12 cases specially examined, the knee-jerks returned in 6 on the day of the crisis, in 4 on the following day, and in 2 one day later. An examination was made of the superficial reflexes, but nothing correspondingly

abnormal was found. Westphal's sign was practically never present in cases of typhoid, erysipelas, measles, scarlatina, and other acute infections. The writer concludes that absence or diminution of the knee-jerks is a very frequent sign accompanying lobar pneumonia in children, and is of great diagnostic value.

J. D. ROLLESTON.

TENDON REFLEXES IN TABES COMPLICATED BY HEMI-
(218) **PLEGIA. (L'État des réflexes tendineux au cours du tabes compliqué d'hémiplégie.)** NOICA, *Journ. de Neurol.*, March 5, 1907, T. xii., p. 81.

WHEN a hemiplegia supervenes on tabes, as a rule the deep reflexes are not increased thereby; occasionally they become so. Examples of both occurrences are cited from the literature, and the two following observations detailed:—

(1) Male, 26. Tabetic symptoms ten years; incomplete, total left hemiplegia seven months. When examined there was just a trace of weakness in the left leg, the hemiplegia having almost completely recovered. All the deep reflexes were absent, except the supinator-jerks; this was normal on the right side, but very exaggerated on the left. The cutaneous reflexes were abolished on the left side only, except the plantar, which shewed a typical Babinski sign on this side.

(2) Male, 43. Some difficulty with micturition for a year, never any other tabetic symptom. Right incomplete hemiplegia a month ago. The cutaneous reflexes were diminished on the hemiplegic side. The left patellar-jerk was normal and the right exaggerated. The only signs of tabes were absent Achilles-jerks on both sides, and Argyll-Robertson pupils. Sensation was natural.

The author concludes that if the tendon reflexes are once lost in tabes they never return; if, however, they are preserved, in however slight a degree, the occurrence of hemiplegia will increase them.

ERNEST JONES.

PSYCHIATRY

THE INCREASE OF INSANITY (The Lumleian Lectures). G. H.
(219) SAVAGE, *Lancet*, March 30, and April 6 and 13, 1907.

THE question of the increase of insanity is one which is at present engaging the serious attention of most civilised countries. According to the British Lunacy Blue-Books, in England and Wales since 1859 the certified insane have increased 231 per cent., as compared

with an increase of 65 per cent. in the general population; in Scotland since 1858 the certified insane have increased 200 per cent., as compared with an increase of 56 per cent in the general population; and in Ireland since 1880, a considerably shorter period of review, the certified insane have increased 77 per cent., notwithstanding a decrease in the general population to the extent of 15 per cent. There are thus ample grounds for the conviction which Dr Savage expresses at the outset of these Lectures, that there is a steady increase of the insane in the British Isles; but he holds that there is no cause for alarm, and gives it as his opinion that this increase is to be explained rather as an outcome of the progress of civilisation than as pointing to an actual spread of brain disorders. "With the multiplication of rules and regulations . . . many cases are attributable rather to social misfits than to material brain disease." The insane include not only many who are popularly spoken of as being "out of their senses" or "out of their minds," but also many who, Dr Savage says, are "out of their surroundings," and owe their position to their surroundings. Insanity is judged largely from conduct or mode of reaction to environment, and similar conduct is, or is not, practically regarded as insanity, according to the conditions. Dr Savage thus, while taking primarily the medical standpoint as to what is meant by insanity, is not satisfied with the purely medical definition of insanity as a disease, and indeed is even inclined to doubt whether the social standpoint is not after all the more practically important of the two, the circumstances and environment having always to be considered.

Having thus indicated his view as to what is meant by insanity, and his conviction that the insane are steadily increasing, Dr Savage devotes the greater part of the Lectures to a consideration of the forms of insanity which in his opinion are increasing, and their causes, which likewise he thinks are becoming more prevalent. The reader must refer for particulars to the original Lectures, which are largely discursive in style, and do not readily lend themselves to condensed statement, Dr Savage relying mainly on observations and impressions derived from his own and others' experience, and also on official statistics in connection with certain points that arise in the discussion. It will suffice to say, as regards the forms of insanity which are being manifested by the ever-increasing insane, that, firstly, "the insanity of brain disease" shows a considerable increase, according to the Commissioners' returns, in each of the three forms—the chronic insanity of confirmed and long-lived asylum residenters, senile insanity proper, and general paralysis, which is referred to as "the insanity associated with premature decay, with toxic brain infection." General paralysis is noted as becoming more common in all

three countries, and also among women in England and more so in Scotland, especially in Edinburgh; and also among negroes in America. Secondly, "the insanity of brain intoxication," comprising psychoses attributed to bacterial infection and characterised by delirium tending towards destruction of mind, may be said to be increasing in the sense that there is at present a tendency in certain quarters to regard insanities, like many other diseases, as toxæmic in nature; but Dr Savage recalls Sutton's note of warning as to over-estimating the importance, as effective causes of disease, of microbes which in health are of use to the economy, but are ready to prey on the latter when it begins to fail in any direction; and later he emphasises the importance of neurotic inheritance in the causation of insanity, and states his belief that a large number of those who are now described as suffering from dementia præcox belong to neurotic stock. Thirdly, in Dr Savage's opinion there is an increase in "the insanity of the truly neurotic type," comprising the insanities usually associated with insane or degenerating parentage, such as melancholia (which is distinctly more common than formerly), mania (especially its severe delirious form, not simple mania which is stated to be decreasing), circular insanity, delusional and hallucinatory insanity, dementia (towards which the insanity of youthful subjects seems to end more rapidly than formerly), and such forms as the insanity of obsession, folie de doute, simple disharmony with the social environment, and functional mental disorders. Dr Savage considers that many of these have merely a dynamic, not a material pathology. No definite opinion is expressed as to any increase of idiocy and imbecility, but it is thought that there is an increase of the minor forms of defect as represented by the growing army of social failures.

As regards the causes of insanity that are thought to be more prevalent than formerly, and to account for the existing increase of insanity, Dr Savage refers specially to three of the principal factors, hereditary predisposition, alcoholic excess, and influenzal poisoning, and would place the last first. Dr Savage believes that influenza acts as an important factor, both predisposing and exciting, in the causation of neuroses, and that "it has been a real and efficient cause for inducing any increase which now exists," and that "it is and has been more powerful than all the other causes put together." It may set up any form of psychopathy or of insanity, though there is no specific form of influenzal insanity. It may also modify an attack of insanity, and, curiously, may even relieve it. Alcoholic intemperance is acknowledged to be a potent cause, not only of actual insanity, but of nervous weakness and instability in the individual and his offspring; but in view of the difficulty of correlating the fact of the increase of insanity with

the recent lowering of the national drink bill, Dr Savage is not inclined to attribute the increase of insanity to any national habit of alcoholic excess. Hereditary predisposition he thinks is on the increase, and finds support for his opinion in the Supplementary Report of the Irish Inspectors who invited the views of the Medical Superintendents of Irish Asylums as to the causation of the increase of insanity in Ireland. Dr Savage does not hold any brief for the hereditary transmission of insanity, but he strongly believes in the transmissibility of the neurotic type which engenders insanity. Dr Savage has also a word to say about certain growing social evils appertaining to both celibate and married life, and their influence, especially as exploited by quacks, in producing youthful hypochondriacs and suicides and neurotic women, and considers that some of the increase of insanity is attributable to such causes. C. C. EASTERBROOK.

MENTAL DISSOCIATION. (Trois cas de dissociation mentale.) (220) AUG. LEMAITRE, *Arch. de Psychol.*, Jan. 1907, p. 252.

1. *Subconscious Inferences.*—A young waitress, separated from her husband, sought assistance in repelling certain dishonourable advances to which she was afraid she would yield. She was easily hypnotised, and suggestion put her subconsciousness effectively on guard.

Deductions: (1) Weak-minded persons may have a subconsciousness alive to danger, but unable to combat it. Lucky if they have the wit to run for help! (2) Indecision is often due to the inability of an upright, but timid, subconsciousness to rise into the field of consciousness without external aid.

2. *Traumatism provoking the Remembrance of an Autoscopic Dream.*—Case of a schoolboy who fell on his forehead and immediately had a repetition of a dream of several nights before, of which he could not have given any coherent account before his accident.

Deductions: (1) Traumatism, dissociating abruptly the superior from the automatic psychical centres, is capable of opening the door to dreams of which not the smallest remembrance remains. (2) External and internal autoscopy are not solely found in hypnotic states, but may also be present in the deep of dreams, the which is a proof the more of Grasset's theory that there exists an automatism high in the nervous scale and functioning after the fashion of the higher psychical centres.

3. *Suicide for Conscience's Sake.*—Case of a clever and neurotic clerk of 18, who had a habit of interjecting into his everyday discourse unfinished phrases which had no connection with the

context of the conversation, and who shot himself with every sign of premeditation after an unjust accusation.

J. ROY TANNAHILL.

TREATMENT.

THE CARE AND TREATMENT OF INEBRIATES. G. BUTLER (221) (of Chicago), *Journ. Am. Med. Ass.*, Feb. 23, 1907.

As a guide in the management and treatment of inebriates, Butler has classified them as follows: (1) Congenital defectives, a type of the pseudo-criminal variety, who use alcohol as a means of shirking responsibility and securing sympathy. (2) Unstable, weak-willed individuals, whose condition is dependent to a large degree upon their environment—easily influenced, often victims of periodical buoyancy and depression of spirits. (3) Accidental inebriates, people who have been uncommonly strong-willed, but have met with some moral shock which has upset both physical and intellectual balance. (4) Hysterical inebriates, closely allied to the weak-willed variety. (5) Periodical inebriates or those who suffer from dypsomania, a condition akin to epilepsy. (6) Those with an acquired nervous constitution—paranoic neuroses, states which follow traumatism, insolation, electric shocks, essential fevers, pelvic disease, and business worry.

There are no drugs which can be employed as specifics in the treatment of inebriety, since it is the patient and not the disease that needs to be treated. The symptomatic use of Strychnine nitrate, Mandragora, etc., as stimulants, and of Sulphonal, Trional, Lupulin, etc., as sedatives is advisable. It is of great importance that the inebriate place himself under medical control for a prolonged period, separating himself from his family and his former surroundings.

The attitude which the public takes in regard to this type of disease is radically wrong. People look upon the drunkard as a good-for-nothing scapegoat; he is denounced by the preachers and punished by the judges, when he should receive the same amount of care and treatment which is allotted to tubercular patients.

Sanitoria or institutions for the care and treatment of these cases should be established and maintained by the State. They should be located in the country, where there is sufficient land to afford work for the patients, thus giving them the benefit of fresh air, sunlight, and exercise. The best medical treatment, baths, etc., should be provided; the discipline should be carried out with firmness and kindness; lectures on this subject should be given for the education and enlightenment of the patients.

The length of time a patient should stay in such a place varies

according to the individual and the duration of the illness; speedy cures are suspicious and almost invariably result in relapse.

C. H. HOLMES.

THE PSYCHIC TREATMENT OF INEBRIETY AND ITS RELA-
(222) **TION TO THE SO-CALLED CURES.** L. MASON (of Brooklyn), *Journ. Am. Med. Ass.*, Feb. 23, 1907.

THE author believes that the numerous remedies which are exploited for the treatment of inebriety should be looked upon in their proper light, viz. as aids through the influence of psychic laws and not through any specific effect of either drugs or "cures." Such influences are active only upon susceptible individuals, and those in the formative stage of the disease. The originators of "cures" make claims which do not show rational therapeutics, and advocate methods which are neither ethical nor humanitarian. The author believes that there are no specifics in the treatment of inebriety in the sense that quinine is given in malaria or mercury in syphilis. In certain selected cases, psychotherapy meets with some success, especially in the earlier stages of the disease.

C. H. HOLMES.

A NEUROLOGICAL CRITIQUE OF RECENT SURGICAL TREAT-
(223) **MENT OF CEREBRAL BIRTH PALSIES.** CHAS. E. ATWOOD (New York), *New York Med. Journ.*, Jan. 19, 1907.

THE clinical manifestations of this condition are medically incurable, and, till recently, the brain injury has been regarded as beyond surgical aid. The cases may be divided surgically into two groups, according to whether they are seen soon after the birth trauma, or only later on account of the paralytic and other symptoms.

1. *Treatment of cases diagnosed soon after birth.*—It was formerly believed that the new-born infant would not stand the shock of a trephining operation, even when a localised hæmorrhage could be diagnosed. Cushing has, however, since 1903, operated upon 8 cases with 4 successes. In his first 2 successful cases the children were respectively 8 days and 7 days old. In view of the serious, often fatal, results of the condition when untreated, surgical interference in intracranial hæmorrhage seems fully justifiable. It would be more universally successful were it not for the fact that the brain structure may be injured simultaneously with the occurrence of the surface hæmorrhage. Even where brain injury exists, relief from the pressure of the hæmorrhage prevents further

secondary destruction of brain tissue. The chief general symptoms are bulging fontanelle, asphyxia, slow pulse, and convulsions; while localising motor symptoms are usually present. Even cases of basilar hæmorrhage may sometimes be improved by a decompression operation over the convexity of the brain. It is said that the presence of red corpuscles in the cerebro-spinal fluid obtained by lumbar puncture distinguishes paralyses due to birth hæmorrhages from those due to prenatal causes.

2. *Treatment of later manifestations of infantile cerebral hæmorrhage.*—Nothing, of course, can at a late stage be done for the actual brain lesion. Retention apparatus, electricity, massage, and education are all useful. Tenotomies and lengthening of tendons may overcome spastic conditions; and muscle and tendon transplantation, or the insertion of the affected nerve into an adjoining healthy trunk, may improve the paralytic symptoms. Prevention is better than imperfect cure, and the conclusion seems to be that surgical intervention in intracranial hæmorrhages of the new-born is, in properly selected cases, a justifiable operation.

W. J. STUART.

A SHORT CONTRIBUTION TO BRAIN SURGERY. (Ein kurzer. (224) Beitrag zur Hirnchirurgie.) EMIL SENGER, *Berlin. klin. Wochenschr.*, Dec. 17, 1906.

1. *Prophylactic Arrest of Hæmorrhage by Osteoplastic Resection of the Skull.*—In cases of fracture of the skull, hæmorrhage from the vessels can sometimes be arrested by the use of suitable artery forceps, though not in all instances. Doyen employed a modification of Esmarch's bandage in his extensive craniectomies. Lothar Heidenhain is of opinion that some bandage of this kind should be applied before the commencement of operations on the head, and has thereby doubtless initiated an advance in the technique of osteoplastic resection of the skull. He places continuous sutures on the inner and outer sides of the projecting section of bone, the sutures terminating at the cutaneo-periosteal bridge of bone. This renders the field of operation practically free from blood. The objections to this method are that it occupies a considerable amount of time, and that the process of suturing causes displacement of the portion of bone from the capsule of the skull, this loosely attached piece of bone being consequently easily detached from the periosteum. In dogs the writer has performed the osteoplastic operation within a single circular or quadrangular series of sutures, the periosteum remaining turned back for an hour, and then fixed with sutures. No necrosis of the skin or portion of bone has resulted. In man he has not ventured to

complete the circle of sutures, but carries them as far as the ridge of bone, leaving a small gap on both sides, or one in the centre. If one considers the course and principal branches of the larger vessels, such as the temporal artery, posterior auricular and occipital, the suturing may be made more or less tense in different parts, in order to ensure better nutrition of the flap. Should the base of the bony flap not be situated in the neighbourhood of the upper and larger vessels and their branches, but only where slender capillaries are found, the gap at the base may be as wide as the base itself. V. Hacker remarks on the advantages of this method in many instances, as the base of the flap cannot only be turned towards the ear, but also in any other convenient direction. In a case of Jacksonian epilepsy, the writer decided to perform this operation under local anæsthesia, by means of alypin and suprarenin. On perforation of the bone a severe epileptic attack occurred, and there was an enormous amount of hæmorrhage from the bone, which did not cease until the end of the attack.

2. *Formation of an Elastic Valve of Bone in Operations for Epilepsy.*—If one believes with Kocher that epileptic attacks are produced by local and general increase of intracranial pressure, it is reasonable to presume that the formation of a functionating valve in the bony skull to equalise the pressure is the best therapy for epilepsy. The following method of accomplishing this is recommended: After turning back a Wagner-Wolf flap, the dura is incised by two oblique sections about 6 centimetres square, so as to form flaps of dura in the shape of an envelope. These flaps are turned back, and three of them united very loosely under the undermined skin, between the periosteum and the aponeurosis, in such a way that they form a kind of pad, the tip of each being fixed by a suture. Then, as recommended by Krause, a strip of bone as wide as the finger is removed from the bony roof at three points, and this reduced plate of bone is laid upon the folded dura, which about fills the gap in the bone. The bony plate thus rests upon a kind of cushion, which prevents firmer adhesion. The flap is now sutured, thus forming a valve in the bone. The writer has learnt from experience and from the researches of Friedrich that the above theory is not applicable to all cases of epilepsy, in spite of the fact that in epileptic attacks pressure in the cerebro-spinal fluid is enormously increased. To those who do not agree with Kocher's theory there is no justification for operative treatment in epilepsy, and still less for the formation of a bony valve; but so long as not only temporary but long-standing benefit follows the procedure in many cases, operative interference will be indicated by the insufficiency of medical treatment in the most severe and hopeless cases of epilepsy. DONALD ARMOUR.

ON PALLIATIVE TREPHINING IN INOPERABLE BRAIN
(225) **TUMOURS TO OBVIATE THREATENING BLINDNESS.**
(Ueber die Palliativtrepanation bei inoperablen Hirntumoren zur Vermeidung drohender Verblindung.) ALFRED SAENGER, *Klin. Monatsblätter f. Augenheilk.*, Feb. 1907.

IN this short address delivered at Stuttgart last September, Saenger lays stress on the importance of the relief of intracranial pressure by trephining, even in cases where there is no hope of removing the tumour. The main portion of the paper consists of short records of nineteen cases in which trephining was done solely with a view to save failing sight. In fourteen of these the immediate results were favourable as regards sight and subsidence of the optic neuritis. In two cases the opening had to be enlarged subsequently so as to allow further escape of cerebro-spinal fluid before any result was produced on the optic neuritis. In two cases death followed the operation too soon for any change to be obvious. And in only one case, that of a basal tumour, was the result bad.

Finkh, in 1904, collected from the literature thirty-one cases where trephining had been done for this purpose, and in thirty of these there resulted a subsidence of the neuritis.

Saenger suggests that the best time for operative interference in such cases is when the vision commences to fail. If the operation takes place later then some optic atrophy is always left. If the tumour is localised he trephines over the site. If no localisation is possible, he thinks trephining over the right parietal lobe is least likely to produce serious complications. In trephining over the cerebellum he lays stress on the importance of allowing some time to elapse between the exposure of the dura mater and its opening. He does not think that any other palliative operation, such as lumbar puncture or puncture of the lateral ventricles, can be compared in efficacy with trephining. LESLIE PATON.

Reviews

NEURONE UND NEURONENBAHNEN. Dr P. SCHIEFFERDECKER. Leipzig: J. A. Barth. 1906. Price M. 11.

THIS work, as stated by the author in his preface, is intended for all who are concerned in the study of the structure and functions of the nervous system. It is divided into three parts. Part I. discusses the structure of the nerve cells and their function and

metabolism during rest and during activity; the structure and probable function of the fibrillæ; their various relationships and the changes which they may undergo in certain pathological conditions; and a consideration of muscle cells and their function during rest and activity. Part II. deals with the theory of the neurone and the connections between neurones and their mode of influencing each other. The writer finds all the objections of Bethe and his school to the neurone theory untenable. Part III. considers the mechanism of the nervous system, including the nature of stimulation and inhibition, the development of nerve paths, the chemotactic influences on the nerve cells, the development of memory, fatigue and recuperation, sleep, and the relation of the blood-vessels to general nutrition.

The first part appears to be of special value to students of neurology, giving as it does a very full discussion of the question of the formation and arrangement of the fibrillæ in the nerve cells, and of the relationship of this to function. The writer insists strongly upon the remarkable observations of Tello on the diminution of the number and superficies of the fibrillæ in the nerve cells of the lizard during hibernation, and the increase in number and fineness of the fibrillæ during the summer period of activity, the inference being that the function of the network is to expose the surface of the plasma which can be acted upon chemically according to the needs of the animal.

The changes which take place in inanition and in various diseases are also considered, and, contrary to many recent writers, the author insists upon the possibility of reconstruction of the neuro-fibrillæ after degeneration. The fibrillæ may be re-formed from the plasma of the cell so long as the nucleus retains or can regain its definite function—a fact which is of the utmost importance in neurology.

It is unfortunately not possible, owing to the limits of space, to enter into a further detailed consideration of the points discussed, but we cannot conclude without expressing our admiration of the philosophical spirit in which the work is written. Study of the book is rendered more easy for foreign readers by the statement of the author's conclusions at the end of each chapter. These conclusions, it must be admitted, are largely theoretical and they ignore much valuable experimental work, a great deal of which has been done in England, but they will still be felt by neurologists to contain much that is very suggestive and helpful.

OUTLINES OF PSYCHIATRY. (**Grundzüge der Psychiatrie.**) Dr FRANZ C. R. ESCHLE (of Sinsheim). Berlin and Vienna: Urban & Schwarzenberg. 1907. Pp. 297.

THE author states in his preface that this work is based on twenty-five years of medical activity, which has not been altogether limited to the study and treatment of mental diseases. His exposition has a certain breadth of view, which is frequently missing in the work of those whose horizon is limited to that of their specialty. To those alienists of considerable experience who, with their general views dating back to their university training, have found themselves somewhat at sea in the changed conceptions of the last two decades, the author offers his work as a help in changing their store of experience into currency. He discusses the reasons why the new groups have been formed.

The book consists of three parts: a general introduction (pp. 1-49), the description of the various mental disorders (pp. 50-242), a discussion of medico-legal questions (pp. 243-268).

In the classification adopted the author follows to a large extent the views of Kraepelin, but he does so in a critical spirit, and with a full appreciation of the difficulties which the acceptance of certain of the clinical groups of Kraepelin presents. With regard to his general psychological conceptions, he constantly refers to his indebtedness to his master, O. Rosenbach. The general introduction consists of a very clear statement of his views on general symptomatology; he criticises the view of those who see in pathological anatomy the final salvation, and emphasises the necessity of detailed character-study of the patient in relation to his environment.

The discussion of the various forms of mental disorder, which takes up the bulk of the book, is very satisfactory, and characterises in a concise manner each individual disorder. The symptomatological pictures are perhaps described rather briefly, and practically no cases are recorded; but the author does justice to the complicated nature of the questions involved, and shows no evidence of narrow dogmatism. In the chapter on manic-depressive insanity, he gives an excellent statement of how the various manias and melancholias have been brought into one group, owing to the prognostic importance of certain cardinal symptoms. He does justice to the variability of the symptomatological picture in this disorder, and to the mixed types. While the author considers the anomaly of mood the primary element in the disorder, he gives no good reason for this view and does not seek to reconcile it with the existence of mixed forms.

For some reason Eschle avoids anything like a thorough discussion of the numerous questions which the formation of the

group of dementia præcox has raised. He contents himself with a summary description of the three forms as defined by Kraepelin, and the chapter devoted to this subject consists of six pages; in Kraepelin's text-book 108 pages are given to this subject.

In the chapter on acute hallucinatory confusion (Meynert's amentia) the author calls attention to the fact that the picture can arise where no exhaustion is established, and insists that it is not essentially an exhaustion psychosis. He makes a separate group of what has been described as the "stuporous form of acute confusional insanity," and describes it under the head of acute dementia ("heilbare Stupidität"). The relation of this picture to manic-depressive insanity and to dementia præcox could only be discussed on the basis of actual cases. Special attention has been given by the author to the psychogenetic disorders, and the chapters on obsessional insanity, hypochondria, hysteria, traumatic neuroses and psychoses, are full of interesting analyses. While he does not refer to the work of Freud and his followers, he emphasises the psychogenetic nature of hysteria; he does not consider hysteria as always a congenital fault in the individual constitution, and thinks that the conception of "congenital psychopathic peculiarity" has been pushed too far. He lays great stress as an ætiological factor on the "influence of the social and family environment, *i.e.* of the education in the widest sense." This eminently practical attitude leads to very valuable remarks on education and treatment. The occupations of the young should have definite accomplishment in view, should not allow the possibility of day-dreaming; the child should be made acquainted so far as is practicable with the real facts of life and learn to find its interest naturally directed to a definite circle of duties. It is not the want of satisfaction of various longings, so much as the want of actual cares and useful tasks, which tend to make old maids, young widows, and childless women hysterical. Education must aim at mental discipline, thought for others, pleasure in the accomplishment of definite tasks. With regard to the traumatic neuroses, Eschle lays great stress on the actual physical result of the trauma, and refers in this connection to sea-sickness and railway-sickness, which he attributes to similar molecular changes; at the same time he fully admits the influence of the emotional shock and other mental ætiological factors.

The medico-legal considerations, which form the third section of the book, are stated in the same clear and succinct manner which is characteristic of the earlier chapters.

The whole book is written in a very agreeable style, and gives in a comparatively short space an excellent statement of the present state of psychiatric knowledge; and although the author lays no great claim to originality, it is obvious that the work is based

on actual clinical experience. To anyone who wishes to familiarise himself with what has actually been achieved in psychiatry, this book can be heartily recommended. C. MACFIE CAMPBELL.

THE DISSOCIATION OF A PERSONALITY: A BIOGRAPHICAL STUDY IN ABNORMAL PSYCHOLOGY. MORTON PRINCE, M.D., Professor of Diseases of the Nervous System, Tuft's College Medical School (Boston). New York, London, and Bombay: Longmans, Green & Co. 1906. Pp. 569. Price $2.80.

STUDIES in dissociated personality are always fascinating, and the very dramatic interest of the cases tends sometimes towards the neglect of a thorough scientific analysis of the phenomena observed. The case of "Miss Beauchamp," which is here presented by Dr Prince, cannot be said to lack dramatic interest; but the study of the psychic mechanism involved has not suffered from the piquancy of the narrative. The author has cunningly interwoven biography and mental analysis, so that at the same time the interest is sustained and important general principles are elucidated; the history of the case is a perfect mine of information with regard to the mechanism of the symptoms in various psychogenetic disorders, and the general value of the book is much increased by a well-arranged index.

The patient was a young lady who came under the author's observation at the age of 23. She presented "neurasthenic" symptoms and definite psychical stigmata of hysteria. The patient was treated by hypnotic suggestion, and during one sitting the patient responded in an entirely novel manner, denied any memory of some recent events, referred to Dr Prince's patient as "she," and showed in attitude and manner a personality strangely at variance with the "Miss Beauchamp" of previous consultations. The latter was quiet, dignified, reserved, and extremely conscientious; the new personality was gay, saucy, mischievous, and without many of the accomplishments of the neurasthenic "Miss Beauchamp." This new personality, christened "Sally," later tended to appear spontaneously, and the periods during which "Sally" was on the stage were blanks in the memory of "Miss Beauchamp." "Miss Beauchamp" would frequently come to herself, to find herself in an extremely awkward predicament due to the irrepressible "Sally." "Sally" formed a distinct personality, with a definite character, trains of thought, memories, perceptions, acquisitions, and mental achievements different from those of "Miss Beauchamp." She not only alternated with the latter, but at times manifested herself as a "concomitant extra-

consciousness," acting as a co-consciousness or sub-consciousness and producing a doubling of the personality. Other lines of cleavage in the personality appeared later under various conditions, but the most important was that which yielded a third personality, which presented frailties of temper, self-concentration, ambition, and self-interest, and which "Sally" called the "Idiot." The life of the patient, who was constantly liable to have her personality split along various lines, was a comedy of errors of a very distressing nature. The author was able to trace the original dissociation to an emotional shock at the age of 18, and concluded that even his original patient "Miss Beauchamp" was really a product of dissociation. After a careful analytical study of the case, he was able to a large extent to reintegrate the dissociated personality, and to produce a personality who was free from hallucinations, aboulia, amnesia, and other phenomena of dissociation and automatism, and who enjoyed better physical health than at any previous period. C. MACFIE CAMPBELL.

UEBER DIE PSYCHOLOGIE DER DEMENTIA PRÆCOX. EIN VERSUCH. C. G. JUNG, Privatdozent of Psychiatry in Zurich. Halle a. S.: Carl Marhold. 1907. Pp. 179. Price M. 2.50.

THE author in his preface to this remarkably suggestive book does not claim to give a complete psychological analysis of Dementia Præcox. He admits that his conclusions are tentative, his arguments perhaps unconvincing; he does not object to his work being considered more of the nature of a confession of faith than a conclusive and rigidly scientific demonstration. It is put forward as an earnest discussion of results based on three years' clinical and experimental work. To students unacquainted with Jung's studies on association, and with the work of Freud, Jung, Bleuler, and Ricklin on psychogenetic disorders, the presentation of the subject in this book must appear revolutionary; it is certainly stimulating.

The author refuses to take a fatalistic view of the behaviour and utterances in Dementia Præcox as absolutely strange and inexplicable, but boldly endeavours to trace the origin of each individual symptom, and to explain in detail the genesis of the individual symptomatological picture. He does not take up the general symptomatology of Dementia Præcox, but assumes a familiarity with the clinical picture; his special question is not the what, but the how.

The book is divided into five chapters, in which the following subjects are discussed: Chapter I., Critical exposition of various theories on the psychology of Dementia Præcox. Chapter II.,

The general influence on mental activity of a thought-complex with emotional value. Chapter III., The influence of the emotional thought-complex on the association of ideas. Chapter IV., Dementia Præcox and hysteria. A parallel. Chapter V., Analysis of a case of paranoid dementia as a paradigm.

Before developing his own ideas, Jung clears the ground in the first chapter by reviewing the views of his predecessors on the nature of the mental disorder in Dementia Præcox. These views have been partly based on theoretical considerations, partly on the results of experimental psychology. Sommer has emphasised the lowered attention, and noted the analogy between the frequent blocking of thought in catatonics and the absent-minded reactions of the normal individual in certain moods. His studies disclosed in the associations of catatonics numerous sound associations, a tendency to the repetition of the same responses, and the abrupt occurrence of eccentric responses in a fairly normal series. Such eccentric responses seem to have the same origin as the sudden occurrence of ideas in the mind of the catatonic, ideas which seem quite out of relation to the trend of thought at the time. A similar phenomenon is also found in hysteria. Vogt attributed the phenomenon of "perseveration" (*i.e.* continued repetition of the same utterance or action) to a narrowing of the field of consciousness, no new content occupying the mind owing to a general lowering of the attention or trend of interest. In this there is an analogy between the hypnotic and the catatonic condition. Masselon, in a thesis on the psychology of Dementia Præcox, also emphasises the diminished attention, the generally lowered synthetic activity of the mind. There result apathy, aboulia, a memory disorder which consists in a capricious inability to recall the desired facts; this capriciousness is considered by others to denote a disinclination to recall them, a form of "negativism."

In this connection Jung remarks that the "I do not know" of the hysterical is frequently difficult to interpret; for it may mean more than a disinclination to touch a distasteful topic, the hysterical disassociation may have caused a real inability to recall the unpleasant memory. Weygandt has described the terminal condition in Dementia Præcox as one of enfeeblement of apperception, which is from another aspect a disorder of attention. Pelletier has studied the association of ideas in Dementia Præcox, and finds a resemblance there to the trend of ideas in reverie; a symbolic trend is frequently present, which denotes a very inferior form of thought.

Jung next refers to Bleuler's views on "negativism," a term used to denote a method of reaction which may arise on many grounds, and which, according to Jung, usually depends upon

certain associations. Bleuler has shown the value of association by contrast as a regulating factor in our mental life; it is the check on our suggestibility, and is the mechanism which prevents precipitate action and allows deliberation. This contrast mechanism is called by him "negative suggestibility." The close connection of the two functions is seen in hysteria in the simultaneous occurrence of suggestibility and contrary auto-suggestions; in Dementia Præcox we find negativism, automatic compliance, and echopraxia intimately associated. The contrast mechanism is an element of such fundamental importance that it has a certain independence; it is closely related to the affective and willing aspect of the personality. A relatively slight emotional disorder may give this mechanism the predominance, and produce negativistic phenomena. In psychasthenics a slight reduction of the general mental energy may produce analogous reactions.

To sum up: these authors consider a lowering of attention or apperceptive enfeeblement as characteristic of Dementia Præcox. On this central disorder depend the occurrence of certain associations, the symbolism, stereotypies, perseveration, automatic compliance, apathy, aboulia, capricious memory, and certain negativistic reactions.

The general disorder may explain the occurrence of these symptoms, but does not answer the further question: What determines in the individual case the special grouping of the symptoms, the peculiar content of the hallucinations and delusions? Various workers have offered partial solutions of this question. Stransky emphasised the poverty of the emotional attitude of the precocious dement, and the incongruity between the emotional reaction and the content of thought. Jung, however, insists that the appearance of incongruity in an emotional reaction may merely be due to our ignorance of the subjective attitude of the patient to certain trends. Stransky has shown that under certain experimental conditions with the attention distracted, the productions of a normal individual may show a marked similarity to those in Dementia Præcox—superficial associations, senseless reactions, repetitions ("perseveration"), senseless neologisms, due to the telescoping of words or phrases ("Kontamination"). A similar telescoping or smelting of whole situations into an incoherent conglomerate also occurs, and Freud in his valuable work on the analysis of dreams has pointed out that in dreams the same smelting of phrases and situations occurs, many of the dream-pictures being explained by the uncontrolled imaginative elaboration of this material. Kraepelin, too, has mentioned the similarity between the expressions in dreams and those in Dementia Præcox. In dreams, symbolism is a marked feature, and naturally so, for

the symbol is an inferior form of thought, and in dreams the apperceptive activity is at its lowest.

A further contribution to the psychology of Dementia Præcox has been made by Gross, who looks upon the disease psychologically as essentially a disassociation of mental activity into several simultaneous trends of thought, which have a certain independence; the breaking through of elements from a disassociated trend into that which essentially continues the identity of the personality is held to explain many of the symptoms, including autochthonous ideas, sudden blocking of thought, catatonic phenomena, etc. Catatonic phenomena are "changes of the will itself through a factor, which is felt as something outside of the personality, and therefore as an external compulsion." He holds that the roots of all automatic phenomena lie in chains of association, which are out of relation to the conscious trend of the individual. He does not discuss what determines which chains of associations, or thought-complexes, are split off. The answer to that question introduces the work of Freud, whose influence on this whole line of study is so important. Freud showed in 1896 that some delusional states are really psychoses of defence, that they arise in an endeavour to banish painful memories, and that in them the symptoms are determined by the content of the banished material; in their genesis such cases are therefore similar to hysteria and the obsessions. When, therefore, there arises mental dissociation as the result of weakened apperception or "lowering of the mental level" (Janet), the peculiar reactions which appear to be independent of and strange to the personality are really determined by the split-off groups of ideas, or idea-complexes. The important question remains: Why does a Dementia Præcox result in some individuals from such a dissociation, and not merely a hysteria? Why in hysteria is the effect so labile, and why in Dementia Præcox is it so stable? In the latter the banished complexes dominate the emotional life, break through into consciousness, prevent the further evolution of the personality; in hysteria the dissociation may be corrected. In hysteria one sees the dissociation result from certain emotional episodes, which, in the majority of people, cause no such reaction, but are assimilated; it is necessary to postulate, therefore, a special reaction type in that individual, the "hysterical constitution." Similarly in the development of a Dementia Præcox, even though one should trace each step in the psychological development, the reason for this special form of faulty reaction to disturbing influences has to be sought. One can understand why the faulty reactions, the hallucinations and delusions, when they do arise, should be of such and such a nature, but why should they arise at all? Having stated the problem in this way, Jung awkwardly and abruptly introduces, as a possible upsetting factor, toxines. His theoretical remarks on

this subject, however, are irrelevant to the main argument, and need not be discussed, although for clinical and therapeutic purposes the question is of crucial importance.

In the second chapter are developed the psychological views, of which hints have been given in the first chapter; and the mental life of the normal individual is discussed in the light of what the study of hysteria and Dementia Præcox teaches. In the psychology of the schools, mental activity is, to a large extent, studied only in so far as it appears purposeful, and to have value according to the traditional standards of value of the environment; the individual, as pruned according to the current taste, and in so far as his activity has social worth, is the main object of study.

Psychiatry has primarily nothing to do with standards of value; it has to study the reactions of the individual in all their fulness, whether relevant or irrelevant to current standards; it has to trace the effect of what the individual tries to submerge as irrelevant or unpleasant, as well as the effect of what he endeavours to make the dominant note of his activity. The processes of normal mental life become distorted in hysteria and Dementia Præcox, but Jung repudiates Neisser's view that the fundamental laws of normal thought can have no applicability to the mentation of the catatonic.

In this chapter the author discusses the action and interaction in mental life of ideas and instincts and memories, which from their emotional value are important dynamic elements. Association experiments have shown that the comparatively indifferent elements of thought are much more under command and at our disposal than those that are not indifferent, but which are connected with some memory, or instinct, or trend of interest that has high emotional value. These complexes are rather disorderly elements in the mental commonwealth, have a certain independence, and, if banished, continually show that their activity has not ceased, but has only become more difficult to trace. The deeper the feeling associated with any complex, the more difficult is it to put that complex aside; the purposeful work of the individual who tries to banish these thoughts for the time, shows by certain indications, such as by peculiar mistakes in speech and writing, by various forms of irrelevant activity, odd reactions, humming snatches of songs, disorders in his responses to the association experiment, how influential these banished thoughts are. The deeper the feeling, the wider are the associations of the complex, and, therefore, the greater the number of occurrences which tend to call it up; if the complex is out of relation to the individual's purposeful activity, he has no longer full command of all his powers; certain elements tend to engross more and more of the mental energy which should be devoted to the purposeful adjustments of a well-regulated life. The best example is perhaps the

infatuated lover. He sees nothing except in so far as it has some relation to the beloved. The merest trifles, if related to his idol, make the intimate personality vibrate; the really important issues of life (according to the standards of others and of himself at other times) become temporarily insignificant. His normal interests fade, his usual activities are diminished. In other words there is "a partial enfeeblement of apperception, with poverty of emotional reaction to all stimuli which are not related to the dominating complex of ideas."

The important thing is that if one were not able to put oneself in the attitude of the lover, his conduct would appear to a certain degree hysterical or catatonic. The normal individual is almost incapable of placing himself in the attitude of the hysterical, where the sensitiveness of the complex is much greater. In catatonia we are at present absolutely incapable of feeling ourselves into the attitude of the patient.

Many other examples of the influence on the individual's general behaviour of definite complexes might be given. The victims of unrequited love have a complex of extreme sensibility, the most neutral incidents being misinterpreted in the light of the complex, in glaring disregard of what common sense would suggest. Prudery is simply one form in which inhibited sexual instincts manifest themselves; a similar manifestation of the sexual complex in the more advanced period of life is the interest in such matters as marriages, births, and scandals.

The sexual instinct must find some expression, direct or indirect; where the direct expression is impossible, the indirect influence is seen sometimes in feverish business activity, daring sports, altruistic activities of special character, artistic achievements. The evidence of a buried complex may be an analomous mood; the man with a secret worry may be known for his jests. A double nature thus tends to develop, the distortion by the complex becoming fixed and representing the usual reaction of the individual, while only occasionally does the deeper basis manifest itself directly and show the truer, more harmonious individual. Where there is such a complex, a single word or gesture may touch the hidden wound. In Dementia Præcox and hysteria the complexes are very sensitive and have wide connections, so that by studying the associations of a series of test-words given to the patient, one can frequently demonstrate the hidden complex.

The manner in which the value of associations is influenced by various complexes is discussed in the third chapter. In previous studies Jung had demonstrated the important results that may be obtained by studying the reactions of individuals to a series of test-words. Such experiments show us within the

limit of the individual reaction the same phenomena which clinical observation shows in a wider sphere. The author had called attention in these experiments to various "complex-indicators," such as long reaction-time, absence of reaction, repetition of the test-words given, etc.; these indicate that the test-word is related to some special complex. A reaction which is influenced by the complex is frequently followed by a very shallow form of association, *e.g.* a mere sound association or grammatical association. The important fact is, that even an exiled complex can to a certain extent influence the value of associations; it can impede mentation (increase of reaction time), arrest the normal mental activity (absence of reaction to the test-word), cause the associative activity to be of a low order (*e.g.* sound association). The influence of a complex is made still more extensive by the fact that even distant resemblances and symbolic likenesses tend to rouse hidden complexes. The symbolic expression of a complex is, according to the author, best seen in the structure of dreams. He gives in great detail the analysis of one dream, in which he traces the apparently fantastic occurrences to definite undercurrents of the individual's daily mental activity. Such interpretation of dreams as is offered by Jung and Freud is extremely suggestive, and obviously contains much that is true; but when it is pushed to the extreme it sometimes appears more ingenious than convincing. Both authors lay great stress on the analysis of dreams, and Jung again and again refers to the detailed analogies between the mechanism of dreams and that of Dementia Præcox; "allow one in dream to go round and act as if awake, there you have the clinical picture of Dementia Præcox." The analogy is not only in the general picture; it is seen in details, such as the peculiar neologisms and paraphasic utterances, in the external projection of mental elements as hallucinations, in the reconstruction of the outer world in the light of subjective needs. To sum up the third chapter: The normal individual escapes from the obsession of some theme by endeavouring to find some other topic of sufficient interest to engross his attention, preferably something in contrast with the first. Where a complex has been banished by means of certain mechanisms of dissociation and substitution, and where it leads to a certain extent an autonomous existence, and thus lessens the amount of mental energy which is at the disposal of the individual in the reactions of purposeful daily life, we have hysteria. Where the complex breaks through and dominates the mental life, distorting it and making impossible the normal adjustments of the individual to his surroundings, we have Dementia Præcox. Such psychological analysis in no way prejudices a toxic theory of the cause of the deterioration.

The parallel between hysteria and Dementia Præcox, which

forms the subject of the fourth chapter, is perhaps the most suggestive part of the book, for it is the analysis of the mentality of the hysterical which has led to the author's conception of the mechanism of the psychological disorder in Dementia Præcox. Throughout this chapter Jung refers to Janet's descriptions of various characteristic symptoms of the hysterical, but he passes beyond the merely descriptive and tries to show the actual mechanism at work. He discusses in detail the analogies between Dementia Præcox and hysteria in the light of the preceding discussion. He compares the emotional dulling in Dementia Præcox, and the incongruity between content of thought and the accompanying affect, with the characteristic "fine indifference" which the hysterical may display in regard to important topics, and with the striking incongruity between thought and emotion seen in cases of obsession with fears (Angstneurose). In such obsessions the affect is found on analysis to be displaced merely and not without foundation, and so in Dementia Præcox the emotional incongruity may be due to the displaced affect of some complex which has to be looked for. There is in Dementia Præcox, as frequently in hysteria, great difficulty in getting into sympathetic touch with the patient, and in the former it is only for short periods that one can hope to succeed, and the possibility of influencing the patient is very slight.

Further analogies are seen in the anomalies of character in the two disorders. In both one may meet marked affectation, with pretentious claims to certain superior attainments; this attitude finds expression in the general behaviour, and in mannerisms of speech, such as the use of imposing neologisms. Other neologisms have their origins in dreams and hallucinations. In the two disorders under discussion the patients may show marked egotism. obstinacy, and want of consideration for others; other cases show a childish behaviour.

Among the analogies in the disorders of the intellect, Jung mentions the narrowing of the field of consciousness, the variations in clearness of grasp, the episodes where the patient feels as if his thoughts were withdrawn, leaving a gap, the transitory impulses, and abrupt hallucinations (sensory automatisms). The disorders of the attention are in the centre of both disorders. The content of the episodes of hallucinatory confusion is in both cases to be finally traced to important complexes. Hallucinations, delusions, and obsessions in both show great similarity in their psychogenetic mechanism, and in their relation to mental undercurrents, which alone explain the nature of the affect. The feeling of vague incompleteness, which the hysterical may have, in perception and volition, has great similarity with complaints in Dementia Præcox, This feeling of inadequacy in perception and volition has its root

in the fact that the dominant complex has withdrawn a large amount of the energy which would normally be devoted to the purposeful receptive and emissive adjustment of the individual to the consecutive demands made upon him by his environment. The obsessions are another form in which an autonomous complex disturbs purposeful thought. Negativism is to a large extent a blocking due to the influence of under-currents; it may be partial or general, and may spread to the motor mechanism.

After a short discussion of stereotypies, Jung sums up the results of the comparison of hysteria and dementia præcox. In hysteria there is at the core an autonomous complex, which tends to disturb the normal associations of purposeful activity, and to weaken the normal adjustment to the environment. In the dreams and deliria of the patient the realisation of hidden longings is enacted. The hysterical may regain equilibrium through assimilation of the complex, which then takes its proper value in the mentality of the individual.

In Dementia Præcox the complex or complexes become fixed and can no longer be assimilated. How far the complex is a causal factor in the genesis of the disease, and how far the disease is of quite different origin although the special features may be due to the fact that a special complex chances to be uppermost at the onset and thus becomes fixed, are questions which await solution.

The detailed analysis of a case of Dementia Præcox, paranoid form, is the last chapter in the book. In some respects it is the most important chapter, because it enables the reader to form a personal opinion on the nature of the evidence which is used to support the conclusions of the author; it is of practical value in so far as it demonstrates his methods of analysis.

The patient was a dressmaker, aged 42 on admission, who presented during a long residence in hospital a typical picture of paranoid dementia. Jung gives her reactions to eighty words used in the association-test and analyses their character; he shows how they betray the influences of various dominant themes, definite trends of interest (Complexes); her delusions were grouped around three main complexes—ideas of ambition and of self-assertion, ideas of being slighted and ill-treated, sexual ideas. The peculiar neologisms of patient (*e.g.* Doppelpolytechnikum) are traced to their origin, and their exact significance in the mental life of the patient estimated; examples of the symbolic trend of thought, as in a dream, are given. The complete analysis shows how the patient, who in her days of mental health had suffered from want and had probably nourished many unsatisfied longings, in her mental disorders constructed a fantastic and complicated picture of life, quite out of relation to the actual facts, but in which, as in

a dream, one could trace detailed effects of the actual hopes and disappointments of her life.

The book is one of extreme fascination; the conclusions of the author may not be accepted in detail, but they are sufficiently convincing to establish the possibility of an analysis of the symptoms of Dementia Præcox much more profound than the Heidelberg school has ever admitted. After reading this book no student can feel satisfied with his examination of a case of Dementia Præcox, unless he has made a much more thorough character-study of the patient than he was previously accustomed to do, and the necessity of this character-study is plainly not limited to the group of the deteriorating psychoses.

The author, not feeling in a position to absolutely establish his position, has allowed himself some of the freedom of a confession of faith; he has made several suggestions as to the actual ætiology of Dementia Præcox, and introduced toxines as allies of complexes in a manner which is difficult even to conceive. These suggestions are irrelevant to the actual psychological analysis, but since the author has introduced the wide ætiological question, one is entitled to bring forward other possibilities than that of a toxine-modified psychogenesis, and to quote the words of Professor Adolf Meyer in his important contribution to the discussion of Dementia Præcox at the Toronto meeting of the British Medical Association, 1906 (vide *Brit. Med. Journ.*, Sept. 29, 1906): "The general principle is that many individuals cannot afford to count on unlimited elasticity in the habitual use of certain habits of adjustment, that instincts may be undermined by persistent misapplication, and the delicate balance of mental adjustment and of its material substratum must largely depend on a maintenance of sound instinct and reaction type. . . . Mind, like every other function, can demoralise and undermine itself and its organ and the entire biological economy, and to study the laws of the miscarriage of its function and life is one of the conditions for any true advancement in psychopathology."

C. MACFIE CAMPBELL.

PHYSIKALISCHE THERAPIE DER ERKRANKUNGEN DER ZENTRALNERVENSYSTEMS, INKLUSIVE DER ALLGEMEINEN NEUROSEN. Bearbeitet von Privatdozent Dr H. DETERMANN. Pp. 126. Stuttgart: Ferdinand Enke. 1906. Price M. 3.

THE change which has within the last generation come over the views of the medical profession in regard to the therapeutic helplessness of cases of nervous disease is due, at least in part, to the adoption of physical, as opposed to medicinal, methods of treat-

ment. In various organic diseases of the central nervous system it has become more and more obvious that many of the symptoms are not the primary result of the lesion, but are attributable to a secondary and functional or nutritional involvement of neighbouring structures. Resort to physical methods has often the effect of limiting the disease to its anatomical site; further, it stimulates the compensatory function of other parts which stand in close relation to the affected part. One of the aims of physical treatment is to revive the stimuli which are constantly passing centripetally from skin, muscles, and sense organs, and so reflexly to raise the tone and stimulate the activity of various important bodily functions, which in their turn have a beneficial effect on the injured nervous system.

From the point of view of physical treatment, one of the first indications in disease of the spinal cord, for instance, is the removal, or at least the neutralisation, of the pathological processes which underlie the morbid phenomena. This indication is often attained by improvement in the blood and lymph circulation, and by regulation of the pressure in the spinal canal. Various mechanical and hydrotherapeutic methods appear to influence the function of the spinal cord in this way. Whether the action of electricity is of this nature, or dependent on chemical effects, is still debatable.

An idea of the contents of this book may be gained from a consideration of the suggested treatment for a case of tabes dorsalis.

Once a diagnosis of tabes is made, the author begins by reorganising the patient's mode of life and finding out whether his occupation or profession may prove detrimental in any way. Every part of the body which is liable to be specially affected must be specially cared for. The bladder must be emptied regularly every hour or every hour and a half. The patient "must not cry aloud, or in any way invite the onset of laryngeal crises." He must avoid tachycardia or faints by not allowing himself to indulge in too much muscular exercise, and by shunning excitement in any shape or form. Every day "he should lie quietly for hours," if possible, for eight hours, and in the open air. The author pays special attention to open-air treatment, and attributes astonishing results to its virtue. He supplies us with details as to the arrangement of the couch in garden, balcony, or verandah; how it must be comfortable and sheltered; how the patient must not have too many clothes on, and yet must avoid catching cold; how he must be able "to busy himself with reading, writing, or eating," just as indoors. Short walks are recommended along level roads or paths.

Leaving general questions aside, and coming to more special forms of physical treatment, the writer extols the value of massage,

exercises, electricity, baths, and hydrotherapy. Even in the preataxic stage, regulated exercises to the limbs are recommended. Cycling in moderation is often a useful adjunct.

For lancinating pains, local hot baths are suggested; galvanism, massage, hot compresses, poultices, etc., may be of service. When they are severe, considerable relief has been obtained by rubbing into the part affected three grammes of "unguentum colloidale Credé" daily for several days. For gastric crises, irrigation of the stomach with hot solutions is occasionally helpful. In cases of bladder difficulty the author has found local massage to the neck of the bladder (one finger in rectum and other hand over symphysis pubis) not without effect. And so on.

Much that is enlarged on in this volume seems so natural to the English mind that one wonders at its being referred to at all; and many points are emphasised which any practitioner of common sense would never think of ignoring. The arrangement of the book by diseases has the great disadvantage that it leads to an inordinate amount of repetition. Thus, under the chapter devoted to the Neuroses, we find the following suggested for the physical treatment of nervous insomnia:—

The patient's mode of life, his daily routine, the relation of work to play, the time of his meals, the quality of his food, must all be attended to. The bed must be comfortable, hard or soft as the individual prefers. The windows of his bedroom should be open during the night, unless noises disturb his sleep. Sometimes it is an advantage to put little "Gummihütchen" in the ears at night to deaden sounds. Climate and locality are important. Innumerable varieties of baths are specified, cold packs and compresses, etc. General massage, gymnastics, exercises, faradic baths, galvanism to the head, etc., are once more mentioned.

Much of the redundant description of the book can be ignored if the practitioner enjoins on his patient the "Leben eines vernünftigen Greises" which the author himself recommends for the tabetic.

S. A. K. WILSON.

ZEITSCHRIFT FÜR DIE ERFORSCHUNG UND BEHANDLUNG DES JUGENDLICHEN SCHWACHSINNS AUF WISSENSCHAFTLICHER GRUNDLAGE, Bd. 1, H. 1, 1906.

THIS new periodical is a central organ for the discussion of the various clinical, anatomical, and therapeutic problems presented by the juvenile weak-minded. In psychiatry the various issues are focussed, and so the list of those associated with the new journal

contains a large proportion of alienists; but this does not mean any intention of minimising the interest of the pedagogue and psychologist, the criminalogist and sociologist in the subject. The journal appears every two months, and promises to be a valuable addition to the present periodical literature.

C. MACFIE CAMPBELL.

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AN INTERNATIONAL CONGRESS OF PSYCHIATRY, NEUROLOGY, PSYCHOLOGY, AND THE NURSING OF LUNATICS

will be held at Amsterdam on the 2nd to 7th September 1907, under the patronage of Her Most Gracious Majesty Queen Wilhelmina and of His Royal Highness Prince Hendrik of the Netherlands. The Congress will be divided into two parts: General Sessions and Meetings of Sections. In the former, chosen subjects will be dealt with by competent men who have been invited for that purpose; in the latter, questions will be discussed and criticism will be invited. Anyone taking an interest in Psychiatry, Neurology, Psychology, and the Nursing of Lunatics may become a member of the Congress. Those wishing to attend the Congress are requested to communicate with the General Secretary, Prinsengracht 717, Amsterdam.

MÖBIUS PRIZE.

The Editor has been asked to intimate that it is proposed by a number of the friends of Dr Paul Julius Möbius, whose recent death the neurological world universally deplores, to found in his memory an annual prize—the Möbius Prize—for a work either on some neurological or some psychiatrical subject. The Committee of Organisation includes the names of Dr Bresler of Lublinitz; Professor Edinger of Frankfurt; Walther C. Jäh, Publisher, Halle; Dr Lamhofer, Ophthalmic Surgeon, Leipzig; Dr Lehmann, Leipzig; and Professor Moeli, Berlin. Further details may be obtained from Herr Curt Reinhard, Lessingstrasse, Leipzig.

Review
of
Neurology and Psychiatry

Original Articles

THE OCCASIONAL LONG DURATION OF BRAIN TUMOUR, WITH THE REPORT OF A CASE OF JACKSONIAN EPILEPSY OF EIGHT YEARS' DURATION AS THE ONLY SIGN OF A SMALL CEREBRAL GLIOMA.[1]

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REMARKS BY DR SPILLER.

THE existence of symptoms of cerebral tumour during a period of eight years, with the finding at the end of that time of a tumour 1·5 cm. by 1·5 cm. by 0·5 cm. is so remarkable that the following case seems worthy of report.

Woods, 60 years of age, entered my service in the Philadelphia General Hospital, June 17, 1904.

On examination after admission he wrinkled his forehead well on each side. The pupils were equal and regular in outline

[1] Read before the College of Physicians of Philadelphia, April 3, 1907.

and reacted to light and in accommodation and convergence. The movements of the eyeballs were good. The tongue was protruded in the median line.

He was seen June 18 by Dr Weisenburg, when the following notes were made: He had been well until eight years previously, at which time on one occasion he felt dizzy, and suddenly had twitching in his left fingers; then the twitching extended to the left shoulder and down the left side. He does not know how much time elapsed before he had a second attack. He had been getting worse during the past four or five years. His mentality is affected and he has some failure of memory. He has had many convulsions during the past four or five years, and the night before admission had convulsions all night. He knows when they are coming on, because he is "worked up" as he expresses it, before they appear. They are confined to the left side. The nurse states that he has had many convulsions since he has been in the hospital, and that they have all been confined to the left side. The patient says that after the convulsions he loses power in the left limbs, especially in the lower limb, and that the limbs feel "numb and dead." The sensation of his left limbs is never normal, and since his first convulsion eight years ago the left limbs have not been so strong as they were previously. He has headache and dizziness when he has convulsions, but never vomits. His sight, he thinks, is good. He denies all venereal infection, and says he drank little.

No impairment of stereognostic perception is detected, and sense of position is normal, as are also sensations of touch and pain in both upper limbs. The left limbs are not wasted. The masseter muscles contract normally.

He was seen by me June 20, when the following notes were made: In a spasm observed by me, both upper limbs became rigid, the face was drawn forcibly to the left, and the eyeballs also were drawn to the extreme left. The eyelids were closed. In about a minute the tonic spasm of the left side of the face and left upper limb gave place to clonic spasms; the left lower limb also was thrown into clonic spasms. So soon as the clonic spasms began on the left side of the body, the right upper and lower limbs became partially relaxed. No clonic spasms were seen on the right side of the body or face. The tongue was not drawn to either side. The attack lasted one or two minutes.

In another attack the spasms were similar to those already described. The eyelids on each side were thrown into clonic spasms and the convulsive movements were equally intense in the lids of each side and in each side of the forehead. There were no clonic spasms of the muscles of the eyeballs. The right side of the lower part of the face was not implicated in the clonic movements.

The wrinkles of the forehead may not be quite so deep on the left side as on the right when the man looks upward. The left upper eyelid is more easily raised by the examiner than the right. When he attempts to smile, the left naso-labial fold is not quite so deep as the right. He does not draw up the left corner of the mouth so well as the right. Sensation of pain is preserved in each side of the face, but the mental state does not permit an examination of the tactile sensation. The tongue is protruded straight, is not atrophied, and shows no fibrillary tremors. The man obeys simple commands and seems to understand what is said to him, although he is not very intelligent, probably because of the frequent convulsions.

The left upper limb is very weak and he is unable to move any part of this limb, but the weakness was not so great yesterday. The biceps tendon reflex is almost abolished on the left side. The left triceps reflex is present, but is not very prompt. Pin prick seems to be felt promptly in the left upper limb. It is impossible to get the patient to fix his attention on the examination of tactile sensation. The movements of the right upper limb are free at all parts, and the grasp of the right hand is good. The biceps tendon and triceps tendon reflexes are not very distinct on the right side. He cannot recognise to-day any objects placed in the left hand, although he recognises them promptly when they are placed in the right hand.

The movements in the right lower limb are good, but the left lower limb is completely paralysed. The patellar reflex is present on each side, and distinctly prompter on the left side. Ankle clonus and Achilles tendon reflex are not present on either side. The Babinski reflex is distinct on the left side, the big toe being moved upward, but is not so distinct on the right side. Pin prick is felt distinctly in each lower limb.

In a third convulsion seen by me, the muscles of mastication on both sides became involved. Five convulsive attacks were

observed during an examination lasting one hour, and they were all of the same character.

Dr Hansell examined the eyes and reported: O.D. Senile choroidal atrophy. O.S. Senile choroidal atrophy; otherwise eyes are in good condition for the age of the patient.

June 22.—The man cannot be aroused, and is said to have a convulsion every few minutes.

He was referred to Dr Edward Martin's service for operation, and on June 23 an osteoplastic flap was raised. The upper line was ½ inch to the right of the mid-line, and extended about 2¼ inches in front and 1¾ inches behind the fissure of Rolando. The base line was about at the upper border of the auricle, and extended one inch in front and one inch behind the end of the fissure of Rolando. The portion of the brain at the anterior part of the opening was paler, and palpation seemed to Dr Martin to show that this portion was not so dense as that more posteriorly, and it was suggestive of a subcortical lesion such as a cyst. This area when explored appeared to be somewhat softened. A small piece of this apparently softened tissue was removed for microscopical study.

The convulsions confined to the left side continued after the operation, beginning within a few hours. From 8.45 P.M. June 23, to 7 A.M. June 24, the patient had forty-one convulsions, confined to the left side. The convulsions occurred frequently during the few days preceding death, and the patient lay in the position of pleurosthotonos, with the body drawn to the left and the eyes partially drawn to the left, even during the intermissions between the convulsions. In the convulsions the abdominal muscles on the left side were in tonic contraction. The man became gradually weaker, and died June 25, 1904, the convulsions continuing until death.

Only a portion of the right hemisphere was obtained at the necropsy. After this hemisphere had been hardened in 10 per cent. formalin, horizontal sections were made through it. The portion which seemed soft at the operation was found to be the seat of a small subcortical tumour, not reaching quite to the surface of the brain (Figs. 1 and 2). This tumour measured 1·5 cm. by 1·5 cm., and 0·5 cm. from above downward. It was situated in the posterior end of the second frontal convolution, and did not extend into the precentral convolution. Microscopical examination showed it to be a glioma.

The continuance of the convulsions, notwithstanding the exposure of the brain by an osteoplastic flap and relief thereby of intracranial pressure, was suggestive of an intracranial lesion causing irritation.

The implication in the convulsions of the muscles on both sides innervated by the facial nerve only in the upper part of the face, and of the muscles of mastication on both sides, is interesting, and is probably to be explained by the more nearly equal innervation from both sides of the brain of those muscles on each side of the body that commonly functionate together. This bilateral implication of the muscles of the upper part of the face in convulsions, otherwise Jacksonian in type, I have observed previously. Interesting also was the deviation of the eyes to the left during convulsions of the left side of the body. In reporting a similar case in 1902, I remarked that "This clinical observation supports the view that the chief centre for conjugate deviation must be near the motor centres of the limbs, because if these centres were widely separated, we should not expect in a case like this, in which the convulsions were confined to one side of the body, to find conjugate deviation of the head and eyes during the convulsions."[1] The present case is valuable because the movements of the eyeballs to the left occurred only during the convulsions, and the tumour was in the posterior part of the second frontal convolution, the portion of the brain believed to be the chief centre for lateral movements of the eyeballs. I have recently, however, had a case in which more persistent conjugate movements of the eyeballs were produced by a hæmorrhage in the parieto-occipital region.

The most interesting feature of this case is the duration of the Jacksonian convulsions during eight years, and resulting from so small a tumour, for it is presumable that the tumour was the cause of the convulsions. We are inclined to give a grave prognosis when a diagnosis of cerebral tumour is made, and properly so, but it is well to remember that occasionally a tumour may grow very slowly, especially if it is a glioma, and may still be small after several years.

A glioma may grow to large size without causing symptoms. Thus in a case reported by Rosenblath,[2] a large, partially calcified

[1] Spiller, *Journal of Nervous and Mental Disease*, 1903, p. 161.

[2] Rosenblath, *Deutsche Zeitschrift für Nervenheilkunde*, vol. xxxi., Nos. 5 and 6, p. 342.

glioma of the left frontal lobe was found, but symptoms are said to have been present only about five weeks, and to have developed after a fall. In another case the symptoms of the glioma had been present only three or four weeks. As Rosenblath says, there are more cases reported of glioma with a long course than with a short one, but those that have existed for years, even decennaries, are such as appear macroscopically as hypertrophied parts of the brain. The soft tumours rich in cells are of more rapid growth.

Within the past few weeks I have seen a case of tumour of the brain which will be reported by Dr M'Connell. Symptoms of brain tumour had been present only about six weeks, and yet at the necropsy a large glioma was found. It had implicated the greater part of the right frontal lobe, and had extended backward into the posterior limb of the internal capsule.

The occasional long duration of brain tumours has been discussed recently by H. H. Hoppe.[1] He says that some cases with unusually long remissions of the symptoms, like Osler's case, finally end fatally, and he refers to the cases reported by Oppenheim, Gowers, Bernhardt, and Russell.

"Psammomata, lipomata, cholesteatomata may attain a certain size and then cease to grow. Cysticerci and echinococci may die and shrink. The observations confirmed by autopsy are exceedingly rare. Nonne was able to find but four cases, those of Bruns, Oppenheim, and Simeon.

"Aneurisms may cease to grow or become obliterated by the formation of a clot, and the brain accommodates itself to the pressure (Oppenheim, Hutchinson, Hodgson, and Humble). Solitary tubercles may undergo caseation, calcification, and then become encapsulated, causing no further damage (Wernicke, Starr, Knapp, Gowers, Babinski, and Sahlberg). These observations are confirmed by the pathological observations of Simeon, who reports an autopsy in which a calcified tubercle was found, which must have been in the brain for thirty years. Kirschberger's case shows how a tubercle may show all the signs of a brain-tumour for a year and a half, the patient recover and remain well for six and one-half years. The autopsy in this case showed that the tubercle had become calcified. Gummata may become absorbed and disappear.

"Long remissions and even permanent disappearance of

[1] Hoppe, *Journal of Nervous and Mental Disease*, Feb. 1907.

symptoms may occur in brain-tumour, as numerous cases on record show. These cases, however, would hardly come under the head of pseudo-tumours, with a complete disappearance of symptoms, because they had been regarded during life as cases of epilepsy of many years' duration, and after death partly or completely ossified tumours had been found as the cause of the epilepsy."

Hoppe thinks that a case that he has observed shows that a pathological mass may be present in the cortex of the brain for forty years and cause no interference with the ordinary daily life of an individual. A man, sixty years of age, died in his service at the Cincinnati Hospital from acute meningitis. He stated that at the age of twenty-one his left arm became paralysed, but that it soon recovered. He had lived the life of a farmer and teamster for the past forty years. With the exception of some weakness of the left arm and a constant headache, he had always been well.

The autopsy revealed a hard, indurated, cartilaginous mass, about the size and shape of a peanut, situated in the cortex at the posterior end of the first frontal convolution of the right side, and pressing upon the ascending frontal convolution. This tumour was partly cortical and partly subcortical, was not firmly adherent, and was surrounded by recent softening. On splitting it open a very hard, stony, calcareous centre, with a hard cartilaginous covering, was found.

The paralysis of motion and disturbance of sensation of the left limb in my patient were probably the result of exhaustion from the convulsions, and these symptoms became more intense as the spasms continued. Death was probably caused by exhaustion. It is remarkable that the motor cortex was not invaded.

Even when the brain was exposed at the operation we could not be sure of the existence of a tumour, although we found an area which appeared to be pathological, and was the diseased cortex over the tumour. It did not seem advisable to excise this, as we could not be certain of the nature of the lesion, but from the later developments it is much to be regretted that the excision was not performed.

Where Jacksonian convulsions are persistent, even though there may be no other symptom, it seems advisable to operate.

We probably run less risk by operating than we do by permitting the convulsions to continue without surgical intervention, although we should remember that occasionally status hemiepilepticus may occur without organic lesion, as emphasised especially by Leo Müller,[1] Nonne,[2] and Bernhardt.[3] In another case, in which Jacksonian convulsions were the only symptom, I recommended operation, and a small tumour was found and excised. It is in cases of cerebral tumour such as these that we may hope the most from surgery, and probably only when a glioma is so small are we able to remove it fully.

Remarks by Dr Martin.

At the time this patient presented himself to the surgical ward for operation his condition was such as to justify no good from a conservative treatment and very little from operation. Since, however, localising symptoms were persistent and very characteristic, since the exposure of the brain can be accomplished rapidly and with little shock or hæmorrhage, an osteoplastic flap laid out by Dr Spiller in such wise as to give free access to the Rolandic area was rapidly turned back. Bleeding from the scalp wound was obviated by an elastic ligature passed around the skull from the forehead to the occiput and secured from slipping by three catgut sutures. The electric osteotome failed to work because of the burning of the fuse. By means of four trephine openings and a powerful set of rongeur forceps the bone was turned back almost as rapidly as would have been the case with the saw and cutting drill. The dura appeared normal and was pulsating. It was turned back, exposing a soft, bleached area merging with the healthy surrounding brain by imperceptible gradations. A portion of this was excised for microscopic examination. The dura was sutured and the osteoplastic flap brought back to position. There was very little bleeding and no appreciable shock as the result of this operation, and the wound ran a reactionless course.

The matter to be regretted is that the operation was incomplete. The neurological localisation was perfect. The seat of

[1] Leo Müller, *Deutsche Zeitschrift für Nervenheilkunde*, vol. xxviii.
[2] Nonne, *Neurologisches Centralblatt*, No. 12, 1906, p. 589.
[3] Bernhardt, *Berliner klin. Wochenschrift*, Nov. 5, 1906, p. 1443.

the lesion was regioned, but because of the lack of definite surface findings and the unfavourable condition of the patient a thorough subcortical exploration was not made. Such an exploration was certainly justified by the symptoms, and in this particular instance it might have resulted in a cure.

EXPLANATION OF PLATES.

Fig. 1. Photograph of a portion of the right cerebral hemisphere. The line *a* indicates the position of the tumour and the level of the horizontal sections shown in Fig. 2.

Fig. 2. Horizontal sections showing the tumour *a in situ*.

THE AMYOTROPHY OF CHRONIC LEAD POISONING: AMYOTROPHIC LATERAL SCLEROSIS OF TOXIC ORIGIN.

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THE palsy associated with lead intoxication, in its acute or chronic form, has long been a familiar condition, to the general practitioner probably fully as much as to the specialist. The drop-wrist of lead palsy, indeed, is perhaps the instance of specificity of symptoms which comes most readily to mind. Further, the muscular wasting of the extensor groups, coupled with the conservation of the small muscles of the hands, is an equally familiar feature in the clinical picture.

Other varieties of amyotrophy in lead poisoning, however, though certainly not so common as that we have just mentioned, have scarcely received the attention they deserve. In few of the accepted English or Continental text-books, with the exception of Gowers' "Manual," is there any adequate treatment of this topic; most are content to note the occasional occurrence of a "shoulder type," or of a "peroneal or anterior tibial type." Gowers (1) attempts a systematised division of lead muscular paralysis into two forms. These may be distinguished as the "degenerative" and the "primary atrophic" respectively. In the former, loss of power precedes wasting; some muscles may be merely weak, and may recover without undergoing atrophy; the reaction of degeneration is commonly present. The usual seat of this variety is the extensor muscles of the wrist and fingers, although the

supinator longus and the muscles of the upper arm, rarely the small muscles of the hand, the long extensors of the toes, and the peronei may be affected. In the latter, primary atrophy occurs specially in the intrinsic muscles of the hand, but is sometimes extensive and irregular in its distribution, affecting many muscles in all four limbs; faradic and galvanic excitability is usually diminished in proportion to the wasting; loss of power and atrophy proceed *pari passu*; and fibrillation is not infrequent. The two forms may be associated, or they may occur separately.

The pathology of these varieties of amyotrophy has always been a vexed question. Rival theories, assigning the condition either to disease of the peripheral nerves or to a change in the anterior horn cells, have each had adequate support. Gowers thinks that the degenerative type is due to acute degeneration of the peripheral nerve fibres, whereas the atrophic form depends on affection of the nerve cells in the anterior cornua. Speaking of intoxications of metallic origin, Marie (2) says: "Les affections medullaires produites par ces agents ne se présentent guère avec des caractères qui les rattachent directement aux grands groupes entre lesquels se partagent les différentes éspèces de la neuropathologie médullaire. Peutêtre cependant une exception pourrait-elle être faite pour certaines formes d'atrophie musculaire au cours du saturnisme, ces formes s'accompagnant de lésions bien nettes des cornes antérieures de la substance grise, et pouvant être rangés dans la poliomyélite antérieure chronique."

It may be taken, then, that the muscular atrophy of lead poisoning is in part at least attributable to the action of the toxic agent on the motor cells of the cord. Whether the effect of that action is limited to the lower neurons is another matter. In a footnote Gowers (3) remarks: "Whether lead has also an action on the upper segment of the motor path, so as to cause weakness without wasting and with an increase in myotatic irritability, we do not know. . . . No distinct sclerosis of the pyramidal tracts has yet been observed, even in cases of long duration." One or two observers, however, have expressed views and recorded instances which scarcely bear this statement out. To these reference will be made later. It is specially to this point that the present communication is directed. During the last two years there has come under my notice more than one case of undoubted lead palsy, in which the amyotrophy has steadily

progressed in spite of treatment, in which, further, there have appeared at a later stage signs pointing unmistakably to a sclerosis of the pyramidal tracts, so that the condition is finally indistinguishable from what is known clinically as amyotrophic lateral sclerosis. It is not, of course, maintained that clinical identity postulates pathological identity; but the view is upheld that, as a result of the continued action of a known toxic agent, a structural alteration is produced in both upper and lower neurons which reveals itself in a combination of lateral sclerosis with muscular atrophy.

For permission to refer to the cases detailed below I am greatly indebted to Dr Ferrier, Dr James Taylor, and Dr Collier, of the National Hospital, Queen Square, London.

CASE I.—C. N., æt. 43, was admitted to the National Hospital under Dr James Taylor on April 14, 1903.

He started work as a compositor when twelve years of age, and continued till incapacitated by wrist-drop eight years ago. He suffered from lead colic also about eight years ago. Three months after this attack of lead colic he noticed that the right wrist was becoming weak, and that he was unable to straighten his hand out to reach for type. Soon after he remarked difficulty in extending his right fingers, and of these the little finger was the first affected. About six months later the left wrist and fingers became similarly affected, and the forearms wasted as the weakness developed. He ceased work as a compositor, but still occasionally had to do with lead.

This double wrist-drop remained practically unaltered for some years, during which time he had neither pain nor paræsthesiæ. Four years ago he was engaged in lifting heavy weights, and was able to perform what was required of him.

Three years ago the weakness and the wasting seemed to have commenced afresh, as he began to notice a certain difficulty in keeping his head erect, and a certain feebleness about his shoulders. During the last eighteen months he has been unable to lift his hands to his mouth. For about a year he has had difficulty in raising his feet off the ground sufficiently to prevent his toes from dragging, and for six months there have been well-marked double foot-drop and corresponding wasting of the legs.

In all the wasted muscles he has noticed quivering or

flickering, and recently he has suffered from painful cramps in the legs. His sphincters are unimpaired, and there is no involvement of face, tongue, or throat, although his articulation has become a little indistinct.

Examination of the patient revealed a condition of profound muscular atrophy.

The shoulders are square and angular, and when he sits up in bed his head falls forward; to attain the erect position again he leans back and then jerks his head up. The only movements at the shoulder that are not exceedingly weak are those of advancing and adduction. Scarcely any movement is possible at the elbows, but flexion of wrist and fingers is good, though their extension is completely lost. The fingers of both hands are contracted into the flexor position. On both sides thumb movements are practically nil; on the right the interossei remain of fair size and strength, while on the left they are atrophic and feeble. The only muscles of the upper extremity which are at all of fair size are the pectorals and the serratus magnus; all the others, including the supinator longus, are in a state of advanced atrophy.

The musculature of the thoracic cage and of the abdomen is not particularly impaired. Both legs are extended in bed, with the feet dropped and inverted. The thighs are well formed and muscular, but there is considerable wasting of the anterior tibial and calf muscles on the right, and of the anterior tibial group on the left. Flexion, eversion, and to a less extent extension, are poor on the former side at the ankle; while toe movements are bilaterally weak, extension being worse than flexion. The patient exhibits a typical steppage gait as he walks, in addition to a certain unsteadiness.

No objective sensory change is discoverable. The biceps and triceps jerks are lost on both sides; the knee and ankle jerks are brisk, and though there is no clonus, a typical extensor response is elicited on the left, and on the right there is a distinct tendency to extension.

The faradic excitability of the wasted muscles is reduced in proportion to the wasting, but all fibres which persist react briskly. No contraction is obtained in deltoid, supinator longus, extensors of wrist and fingers, thumb muscles, and other of the severely or completely wasted muscles. Similarly, galvanic

excitability is reduced in proportion to the atrophy, and lost in the most affected muscles; no polar change has been noted.

CASE II.—E. B., æt. 32, was admitted to the National Hospital under Dr Ferrier on November 8, 1905.

For the last sixteen years he has been employed as a tinsmith in the works of Vickers, Maxim & Co., and though there is a good deal of lead in the solder used by him, he has never suffered from lead colic.

Two years before coming under observation he began to notice an occasional cramp in the fingers of the left hand, usually in the morning. They seemed to flex involuntarily, and he was unable to straighten them for a moment or two. About a month later, the fingers became rather suddenly weak, and he had difficulty in extending them; at the same time there was some tenderness over the outer aspect of the forearm at the elbow. In the course of a week or two he was unable to hold the fingers and hand extended for longer than a few seconds, and six months after the onset the drop-wrist on the left side was complete. During this time he frequently noticed fibrillation in the muscles above the elbow on that side, but no wasting. A year later, he began to be afflicted with cramps in the right hand, chiefly in the two ulnar fingers, which continued for a month or two, and were followed by a gradual drooping of the fingers and wrist; in about a month the paralysis was complete. In spite of treatment the condition has become worse; about a year ago he noticed muscular wasting in the left forearm, and subsequently in the right; the cramps, however, have been less frequent and less painful.

Three months ago he found that the toes on the right side were beginning to drag as he walked, and that the right leg was becoming weak at the knee. He has not been aware of any change in the left leg, but for at least twelve months he has suffered from very severe cramps in the calves of the legs and in the toes, especially on the right side.

For about two months he has remarked some wasting of the muscles of the right leg below the knee, and for the same time there has been considerable hesitation in micturition. There has been no difficulty in speaking or swallowing.

The first point one noticed on examination was the pallor of

his skin, and to a less extent of his mucous membranes, and there was a distinct suggestion of a "lead line" on the gums. His face was rather expressionless, without any obvious paresis. There was complete double drop-wrist, which he was entirely incapable of correcting; he could extend neither wrist nor fingers, nor could he extend the former when the fingers were flexed. The flexors of wrist and fingers were relatively well preserved; in the effort to grasp there was marked overaction of the flexors, but if the wrist were passively extended the grasp at once improved. There was no flattening of the thenar or hypothenar eminences, no guttering of the back of the hands. If the hand was flattened out it was found that the interossei acted with fair strength. Any movement of small muscles, such as the approximation of thumb and first finger, was accompanied by marked flexion at the wrist.

In addition to the drop-wrist, the patient showed considerable weakness and wasting of the biceps, more particularly on the left side, and also of the supinator longus, especially on that side. Otherwise the arm muscles seemed good, while there was no defect of the musculature of thorax and abdomen.

The right tibialis anticus was somewhat wasted and very weak; the whole of the right thigh was slightly shrunken compared with its fellow; there was a corresponding weakness of extension at the right knee. Dorsiflexion of the foot was poor on the right side, fair on the left, but the toe movements were well preserved. No objective change in cutaneous sensation was discovered. Deep reflexes were present in the upper extremities, and brisk; the knee and ankle jerks were likewise brisk, and though there was no ankle clonus, a typical double extensor response was elicited. The abdominal reflexes were active.

In all the wasted muscles faradic excitability was diminished, and galvanic excitability similarly; it is true, in the case of the wasted extensors, that the galvanic response was not so feeble as in the other affected groups, but there was no reaction of degeneration.

This same patient came under observation fifteen months later, when it was seen that the condition had steadily progressed. The wasting and weakness of the extensor muscles were profound; in addition, the biceps and triceps and supinator longus were all weaker and thinner; the long flexors had become weak; the

FIG. 1.

FIG. 2.

interossei also were affected on the left side, though the muscles of the thenar and hypothenar eminences remained good. There was great weakness of the whole of the right leg, which the patient was unable to lift off the bed; at the knee both flexion and extension were very feeble, and there was considerable shrinkage of the musculature of the right thigh; at the ankle dorsiflexion on both sides was limited, and on both sides flexion and extension of the toes were definitely impaired. Frequent fibrillation was observed in the atrophic muscles, and the patient complained still of constant, painful cramps, especially in the calves. The deep reflexes were still brisk, except where the muscles were greatly wasted, and a double extensor response was elicited as before.

CASE III.—T. B., æt. 37, was admitted to the National Hospital under Dr Hughlings Jackson on September 28, 1904.

He had worked as an engine-fitter for years, but had not, apparently, come into direct contact with lead. Twelve years ago he contracted syphilis. Eighteen months ago he began to notice numbness of the right middle finger, followed by a loss of power in that finger, so that it dropped down and could not be extended. The loss of power in straightening the fingers gradually spread to the others, and at the end of a year he was unable to extend them and the wrist, and began to notice a certain amount of wasting of the forearm. At the same time the right elbow and shoulder became somewhat weak.

Three months ago he remarked a weakness of the left middle finger similar to that which had affected the right, and this was succeeded by rapid loss of power in flexing the elbow and complete wrist-drop. The left shoulder also began to show signs of weakness. Three weeks ago he found that his head was falling forward as he walked, and he had to hold it up with his hand. He has had increasing difficulty in passing water for at least three months.

On his admission to the hospital it was remarked that the patient was pale. The pupils were small and showed the Argyll-Robertson phenomenon on both sides. The tongue was tremulous and appeared slightly atrophic on the right side. All the deep posterior muscles of the neck were weak. The arms were markedly atrophic, and the wrist-drop was complete. On the

right there was great wasting of the pectorals, triceps, supinator longus, and long extensors, with slight wasting of the abductor indicis. The muscles of the thenar and hypothenar groups, however, were quite good. On the left, trapezius, deltoid, biceps, extensors of wrist and fingers were wasted or wasting, but the small muscles of the hand were entirely exempt. In the legs no particular muscle or group of muscles was affected, but the limbs were slightly spastic. All voluntary movements in the lower extremities were well executed. Frequent cramp-like pains in the legs, sometimes shooting in character, were complained of, but no objective change in sensation was found. The triceps-jerk on the right side and the biceps-jerk on the left were diminished or absent, but both knee-jerks were brisk, and a double extensor response was obtained. In all the affected muscles faradic excitability was diminished in proportion to the wasting, and similarly with galvanic excitability.

CASE IV.—E. W., æt. 63, was admitted to the National Hospital under Dr Collier on December 12, 1906.

Since the age of sixteen he had been employed as a painter and paperhanger, and three years ago he had an attack of lead colic. In the beginning of August 1906 he caught a chill when at work, and began to suffer from sciatica in the right buttock and thigh, a "burning pain" which went down to the heel and left the latter numb. He began to notice "flickering" in the muscles on the back of the thigh. About three weeks later he began to suffer from cramp in the fingers of both hands: they used to become bent and stiff, and he was unable to straighten or to separate them. A week later the hands had begun to drop slightly, and the condition steadily continued. Not long afterwards it was remarked that he was becoming pale. There has been no amelioration of the condition; the hands and fingers have become so flexed that he is unable to perform any fine movements with them; he has not, however, thought that there was any weakness of other muscular groups. Just prior to his admission, severe cramps in the legs began to develop. He has noticed fibrillation in the affected muscles.

On examination we find very considerable weakness and wasting of deltoids, pectorals, biceps, triceps, trapezii, and spinatus muscles, and to a less extent of latissimi and serrati. There

FIG. 3.

FIG. 4.

FIG. 5.

is complete double wrist-drop, with contracture of the flexor groups, but no obvious impairment of interossei and muscles of thenar and hypothenar eminences. The legs are well preserved and slightly spastic; possibly dorsiflexion at the ankle is a little less good than might be expected. In the arms direct muscular irritability is increased, and occasional fibrillation is seen. No objective loss of sensation is noted, although the patient complains of aching and stiffness in the arms, and of cramps in the arms and legs. All the deep reflexes are exaggerated; there is ankle clonus on the left side, not, however, well sustained; the plantar response is very doubtful; it is never typically flexor, and occasionally there has been a more or less definite extensor response on the right side. The sphincters are unimpaired. The electrical reactions show a diminution to the faradic current in all the affected muscles, and a more or less corresponding diminution to galvanism. A reaction of degeneration is not obtained.

If we resume the salient features of these four cases, we note:—

1. In each there is the same unmistakable (except, perhaps, in Case III.) source of poisoning, viz. lead, the action of which has been continued over a considerable period.

2. In each the onset is characteristic of an ordinary lead palsy, viz. double drop-wrist. The degree and duration of this lead palsy before the appearance of other symptoms vary greatly in the different cases.

3. The lead palsy, which is possibly of peripheral origin, is followed by a progressive amyotrophy of irregular distribution, affecting flexors as well as extensors, but respecting the small muscles of the hands, at the commencement at least. One of the cases has not, up to the present, showed any wasting of the small hand muscles. The amyotrophy is associated with cramps in the limbs, fibrillation in the diseased muscles, sometimes with involuntary spasmodic movements, and with weakness proportionate to the wasting—all of which symptoms are characteristic of central as opposed to peripheral changes. In two cases evidence is forthcoming of the spread of the disease to the medulla.

4. In all the cases the deep reflexes are exaggerated in the legs, and in some also in the arms; in three we get a double

extensor response, in two ankle clonus, and in three, further, a certain amount of spasticity in the lower extremities. These symptoms furnish indubitable evidence of a varying degree of lateral sclerosis.

5. The electrical reactions argue in favour of a central as opposed to a peripheral lesion.

6. In none of the cases is sensory impairment conspicuous or even discoverable.

These cases thus suggest an interesting line of inquiry. It is, I think, clear that at least two of them are clinically indistinguishable from amyotrophic lateral sclerosis; as a matter of fact, that diagnosis was made, and rightly made, in these two instances, on the patients' admission to hospital. If we except the mode of onset, the two conditions are identical. The absence of any definite sensory changes, apart from paræsthesiæ, the cramps, spasms, fibrillation, the progressive amyotrophy, the electrical reactions, exaggerated reflexes and extensor responses form a picture which differs in no essential detail from the classical Charcot's disease. The fact that in this toxic variety of the disease the small muscles of the hand are at first exempt is no reason for differentiation; we are simply dealing with a type which happens to be somewhat rare, in which the extensor muscles of the wrist and fingers are selected by the morbid process, the small hand muscles being involved later, as in Cases I., II., and III.

Unfortunately it is impossible to find support for this view of the cases by an appeal to the results of post-mortem examination, as the opportunity has not been afforded me. Case I. has been dead for some years; Case II. is steadily becoming worse—the patient thinks his speech is not quite so clear as it used to be; Case III. is probably dead too, efforts to trace him being unsuccessful; Case IV., in spite of long hospital treatment, has made little if any real improvement. One cannot but be struck with the progressive nature of the affection, which is so contrary to the ordinary forms of lead palsy, where cures are by no means unknown. In default of pathological information, the clinical evidence is still strong enough to justify the argument advanced in this paper, apart from analogies drawn from other conditions to be noted immediately.

As far as I have been able to discover, the possibility of

lateral sclerosis in lead intoxication has not been much alluded to, and, with the exception of certain cases quoted by Putnam, I have not found a record of cases comparable to those here given in detail. Gowers cites one or two instances of progressive lead amyotrophy, and speaks of their analogy to progressive muscular atrophy, but in spite of the fact that he considers the latter synonymous with amyotrophic lateral sclerosis—to enter into this question is beyond the scope of the present communication —he does not accept the evidence for involvement of the upper neuron. Moreover, he says that the extensive primary atrophic variety usually begins in the small muscles of the hand, whereas the type now described is an extensive atrophic variety which begins as an ordinary lead palsy.

In a suggestive article written twenty years ago (1887), Putnam (4) alludes to the want of general recognition of the fact that lead poisoning may give rise to symptoms of spastic paraplegia, inasmuch as current clinical teaching was under the influence of the tradition which ascribed lead amyotrophy to peripheral lesions. Brissaud stated that in toxic paralyses the deep reflexes are habitually diminished or lost. Putnam collected eighty-six cases where lead was known to have been the active agent in producing symptoms, and among these he formed a group where examination suggested the presence of a varying degree of lateral sclerosis. One or two may be briefly referred to.

1. Spastic paraplegia; cramps and twitching of the calves; knee and ankle jerks exaggerated; history of chancre; lead found twice in the urine, and a considerable quantity also in the drinking water.

2. Marked progressive spastic paraplegia, with myosis and loss of pupillary reaction to light; ataxia and some atrophy of the hands.

3. Gradual loss of power in extensors of fingers and thumb of left hand, steadily increasing; similar condition in muscles of right hand; tremor on attempted extension of the fingers; knee-jerks exaggerated, trace of ankle clonus; patient says his legs feel stiff.

It is well to remember that these cases were examined long before the diagnostic significance of the plantar reflex was known to the neurologist, but they surely justify the opinion which the

author expresses: "I am by no means prepared to assert that a clear case is made out for the causation by lead of typical chronic spastic paraplegia. I would only claim that the cases I have reported indicate that as a direction in which to look in the future. . . . If it can be shown that lead poisoning leads to the symptoms of spastic paraplegia or to some forms of it, especially those which are somewhat irregular in their clinical history, and are associated with signs of peripheral neuritis on the one hand, and with more or less disorder of the general nutrition of the body on the other, we shall have made a practical gain in our knowledge of a distressing disease."

The clinical documents at our disposal, then, warrant the statement that one of the actions of lead, if it be present in the system for any length of time, is not merely to effect a change in the neuron from anterior horn cell to periphery—a motor "neuronitis," to borrow Grasset's term—but also to leave its trace on the upper neuron, perhaps in its distal section first, wherefrom we derive the characteristic symptoms of spasticity in varying degree, exaggeration of deep reflexes and extensor responses. There is no *a priori* reason why the pyramidal tracts, though not forming a part of the lower nerve segment, which is the first to suffer in lead intoxication, should not be secondarily involved; indeed, there is no reason why they should not be separately attacked.

An interesting and important analogy suggests itself from the effects produced by other recognised toxic agents on the central nervous system. Ergotism, lathyrism, and pellagra are diseases where systematised degeneration of various spinal and other tracts follows the introduction of a toxic substance into the organism. The lesions thus determined present both the clinical and the pathological aspect of certain common affections of the spinal cord; syphilis itself is a toxic agent which leads to a form of spinal sclerosis; and however little understood may be the actual mechanism of the change in nerve elements, there is no valid reason for endeavouring to separate the metallic from the vegetable poisons in their action on the nervous system. In what is known as sub-acute combined degeneration of the spinal cord we have a disease in all probability of a similar origin, though it is not so strictly a system disease; and as the toxin of subacute combined frequently influences the blood as well as

nervous structures, so lead frequently disturbs the function of the bone marrow as well as of the nervous system. As for the pathogeny of the change, it is unwise to speculate where pathological confirmation is awanting; it may, however, be remarked that there is little to favour the view of an ascending neuritis, since Sicard (5) has conclusively demonstrated the rarity, indeed the problematical nature, of that morbid process. The instances that have just been quoted render the theory more tenable that lead may act, though unequally, both on the upper and the lower segments of the cerebro-spinal motor chain.

As regards amyotrophic lateral sclerosis itself, when Professor Marie (6) says of that disease: "Sous quelle influence survient ce processus morbide? On l'ignore entièrement. S'agit-il d'une sorte de dissolution spontanée d'un ou de plusieurs systèmes anatomiques; ou bien l'altération de ces groupes de cellules nerveuses tient-elle à une influence générale? Autant de questions actuellement insolubles"; and again, "Quant à l'étiologie, les renseignements sur les causes de la sclérose latérale amyotrophique font absolument défaut"; it is at the least interesting to find two typical clinical instances beginning with double wrist-drop under the influence of lead.

There are one or two minor points arising out of the cases which may be shortly alluded to.

Case III. was at one stage diagnosed as tabes dorsalis, and this can be readily understood when it is remembered that the patient suffered from lancinating pains and exhibited the Argyll-Robertson pupil. It is true there was a history of previous infection, and one might be content to attribute these symptoms to the usual cause, but for the fact that either may occur apart from preceding syphilis. The pains of lead toxæmia are sometimes very severe; one of Putnam's cases (not referred to above) had marked tremor of the hands and lightning pains in the legs, which it is impossible to distinguish from the pains of tabes. The majority of lead cases are afflicted with cramps in the muscles, more or less painful, and, on the other hand, I have been struck with the frequency of painful cramps in amyotrophic lateral sclerosis, a very large proportion of cases exhibiting this symptom, sometimes early in the course of the disease. The similarity is worth noting. One is reminded in this connection of the condition described as "pseudo-tabes," of which less is

heard now than formerly. The cases originally classified as pseudo-tabes were the result of chronic arsenical poisoning, and were characterised by absence of deep reflexes, ataxia, and lightning pains. If a chronic toxæmia, therefore, can present so close a resemblance to a common spinal cord affection, we can understand that in chronic plumbism the same process is possible. Redlich (7) has described a case of chronic lead poisoning clinically indistinguishable from tabes, and some others have been recorded. In fact, it may be that many system diseases of the brain and spinal cord can thus be brought into line, and their pathogeny elucidated. According to the specificity of the toxic agent a sensory or a motor neuronitis may result, or a mixed neuronitis; and this neuronitis may have a greater incidence on the upper or the lower path.

In Case III., further, the Argyll-Robertson pupil was present on both sides. Here again there would be no call for remark, were it not that the Argyll-Robertson pupil cannot be taken as pathognomonic of preceding syphilis, *pace* the French school. One of Putnam's cases of lead intoxication had fixed pupils, with no history of preceding syphilitic infection; and again I have seen the condition in amyotrophic lateral sclerosis. Within recent years at least two other cases of that disease have occurred at Queen Square where the sign was present, developing in the course of the disease, apart from any question of syphilis. This is, once more, a point worthy of note. It would seem to show that the line of demarcation between the effects of avowedly toxic states and of other disease-producing agents is very difficult to draw. Examination of the mid-brain in amyotrophic lateral sclerosis by Marchi's method reveals definite involvement of certain tracts, such as the posterior longitudinal fasciculus, apart from profuse degeneration. In some cases of lead poisoning the cranial nerves have not escaped, and in one or two recorded autopsies of lead cases degeneration in the brain-stem has been noted.

The occurrence of the Argyll-Robertson pupil in amyotrophic lateral sclerosis is therefore of some significance, as it means that what is admitted to be a sign of toxic activity in one disease may be found in another disease the cause of which has hitherto been entirely unrecognised. Bearing in mind, then, the various points raised by a consideration of the

cases here reported, we do not assert that a *prima facie* case has been made out for the toxic origin of amyotrophic lateral sclerosis, but we cannot avoid the conclusion that the facts suggest the probability of such an origin.

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3. Gowers. *Loc. cit.*, p. 880.
4. Putnam. *Boston Medical and Surgical Journal*, July 28, 1887, p. 73.
5. Sicard. "Le syndrome de la névrite ascendante," Congrès de Rennes, 1905.
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Explanation of Plates.

Fig. 1.—Case III. The photograph was taken on a dull December day, when an instantaneous exposure was impossible, and it shows how the head was falling forward. There is great atrophy of shoulder girdle and other muscles.

Fig. 2.—Case II. Wasting of the extensor groups, with a thin and weak supinator longus.

Fig. 3.—Same case. To show the atrophy of the arms and drop-wrist, with relative conservation of the small muscles of the hands. Weak long flexors.

Fig. 4.—Case IV. Double wrist-drop, with contracture of the flexors.

Fig. 5.—Same case. To show increase of flexor action in the attempt to extend fingers; conservation of small muscles of hands.

FURTHER BACTERIOLOGICAL AND EXPERIMENTAL INVESTIGATIONS INTO THE PATHOLOGY OF GENERAL PARALYSIS AND TABES DORSALIS.

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and
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In previous papers (1) we have recorded observations on the ground of which we have contended that a diphtheroid bacillus, having cultural and morphological characters resembling those of the Klebs-Löffler bacillus, plays the chief part in the production of the toxæmia of general paralysis. We have at the same time insisted upon the importance of weakening of the

local and general defences against bacteria, and have attributed the chief part in the production of this impairment to syphilis, chronic alcoholic intoxication, and the excessive use of nitrogenous foods. A little more than a year ago we thus summarised the case in support of the diphtheroid hypothesis :—

The evidence that a diphtheroid bacillus—either an attenuated form of the Klebs-Löffler bacillus or more probably an altogether distinct micro-organism—is the specific etiological factor in general paralysis and tabes dorsalis is briefly as follows. A bacillus of this nature is, according to the results of our investigations, present in large numbers, either in the alimentary or respiratory tract, or in both, and in the genito-urinary tract, in all cases of advancing general paralysis. This bacillus has a thread form, which has been found invading the walls of the respiratory or alimentary tract in five cases of general paralysis. It can be shown that this bacillus invades the pulmonary tissues in cases of general paralysis, and that it is commonly the only micro-organism present in large numbers in the catarrhal pneumonic foci that occur in most of such cases dying in congestive attacks. A growth of a diphtheroid bacillus has now been obtained in cultures made from the brain post-mortem in ten cases of general paralysis out of twenty-four in which cultures were made from this organ. Diphtheroid bacilli exhibiting metachromatic granules in Neisser preparations have been detected in the fresh blood in one case and in sections of the brain in two cases. It has been ascertained by experimental methods that these diphtheroid bacilli in contact with the living blood are rapidly taken up by the polymorpho-nuclear leucocytes, and that they may be completely digested in the course of two or three hours. Bodies exactly corresponding in appearance to these dissolving bacilli can be detected in the blood and cerebro-spinal fluid of the living general paralytic, especially during a congestive attack. Whilst the fact that most of the bacilli present are in process of disintegration satisfactorily explains the long succession of negative results of endeavours to obtain cultures from the blood and cerebro-spinal fluid, we have, by the use of special methods, succeeded in obtaining pure growths of a diphtheroid bacillus from the fresh blood in four cases of general paralysis, and from the cerebro-spinal fluid withdrawn by lumbar puncture in two cases. In sections of

the brain prepared by special methods, disintegrating diphtheroid bacilli can be recognised in the walls of the vessels and in the pia-arachnoid in many cases of general paralysis. The centrifuge deposit from the urine of the general paralytic, especially during a congestive seizure, commonly contains abundant diphtheroid bacilli that have been more or less affected by lysogenic action. In seven consecutive cases of general paralysis combined with tabes we have found the centrifuge deposit from the urine to contain, not only these altered diphtheroid bacilli, but also living ones, showing distinct metachromatic granules. In such cases a culture of the bacillus can be obtained from the urine. Experimental infection of three rats and a goat with diphtheroid bacilli isolated from a case of general paralysis has resulted in the production of symptoms and tissue changes resembling those of general paralysis. Lastly, there is evidence that the active polymorphonuclear leucocytes of the general paralytic have, as a rule, a greater power of dissolving these diphtheroid bacilli than that possessed by the normal leucocyte. It would therefore appear that the general paralytic has acquired against these diphtheroid bacilli a certain degree of specific immunity, by means of which he is enabled to maintain the struggle against these bacilli, notwithstanding an otherwise defective local and general power of resistance.

These views have been opposed in this country by Eyre and Flashmann (2), Mott (3), Ferrier (4), Bulloch (4), Hamilton Marr, and several of the medical journals. On the other hand, there have not been wanting those who have recognised that the various points of evidence we have brought forward are entitled to consideration, and some have accepted the conclusions that we have drawn from them. As regards general paralysis, many of our observations have been confirmed by those of O'Brien (5) and Langdon (6) in America. O'Brien's work has been extensive, and in respect of his experimental observations he has gone ahead of us. He has isolated an organism resembling the Klebs-Löffler bacillus from 95 per cent. of cases of general paralysis. He has found that lower animals inoculated with the bacillus have passed through attacks similar to congestive seizures, hemiplegia, and objectively typical paresis. Post-mortem examination revealed conditions similar to those seen in an early

case of general paralysis. He produced a bactericidal serum in the goat, and found that it had a protective action upon dogs infected with the bacillus. More recently he has treated cases with vaccines consisting of the killed cultures, using the opsonic index as a guide, and has had a considerable measure of success.

The further observations that we have now to record concern chiefly the broth reactions and virulence of a series of diphtheroid bacilli obtained from various sources, the experimental production of general paralysis in rats, the experimental and clinical study of intra-corpuscular bacteriolytic indices in relation to certain species of these diphtheroid bacilli, and also some special phenomena that bear upon the views maintained.

In 1903, when we first advanced the diphtheroid hypothesis of the etiology of general paralysis, we provisionally regarded the organism as an attenuated form of the Klebs-Löffler bacillus, in accordance with the authoritative teaching of the time that an organism differing from the diphtheria bacillus solely in its want of virulence must be regarded merely as a diphtheria bacillus in an attenuated condition and should be spoken of as such. In taking up this position we left it an open question whether the bacillus might not be a special one differing from the bacillus of acute diphtheria in certain respects afterwards to be determined. A year ago, though we could not absolutely distinguish the cultural and morphological characters of the organism from those of every other species of the diphtheroid bacillus, we felt justified, in view of additional evidence we had collected, in maintaining that the organism is a special one, and we proposed to refer to it as the *bacillus paralyticans*. It was suggested to us by Dr M. H. Gordon that we might be helped in the differentiation of this organism from other diphtheroid bacilli by applying litmus broth tests to all of the organisms of this nature that we isolated from time to time in the course of our investigations. We would take this opportunity of acknowledging our great indebtedness to Dr Gordon for suggesting the application of these tests, and also for other valuable advice regarding our special bacteriological investigations. We have employed Dr Gordon's method, and have used as test substances glucose, saccharose, lactose, salicin, starch, and dextrin. The following table shows the reactions previously ascertained to be given by various of the already known members of the diphtheroid group to the first five of

these substances. The results are those obtained by Dr Gordon, supplemented by some ascertained by Benham (7). The reaction of each organism to Neisser's method is added.

Bacillus.	Glucose.	Saccharose.	Starch.	Salicin.	Lactose.	Reaction to Neisser's Method.
Klebs-Löffler .	+	−	−	−	−	Positive
Hoffmann's b. .	−	−	−	−	...	Negative
Xerosis b. .	−	−	−	-	...	Negative
Diphtheroid x.	+	+	−	+	...	...
Diphtheroid y.	+	+	+	−	...	...
Bacterium muris (Klein)	+	+	...	+	...	Positive
B. coryzae segmentosus .	+ or −	+ or −	...	...	+ or −	Negative

The sign + indicates that a distinct acid reaction is produced by the organism after incubation at 37° C. for three days.

The results obtained by Knapp (8) and Graham Smith (9) with the media of Hiss differ as regards the above test substances only in certain points that are hardly material for our present purpose. It is, however, to be noted that both state that an acid reaction is produced in dextrin broth within seven days by the Klebs-Löffler bacillus.

It has not been possible for us to make any very extensive investigation into the broth reaction of the diphtheroid bacilli that can be isolated from general paralytics and tabetics. We have simply tested with what appeared to be the most important substances the various diphtheroid bacilli that we have happened to obtain in pure culture in the course of our recent work. The following table shows our results up to the present date. All of the strains gave a positive reaction with Neisser's method, but in the case of numbers 9, 10, and 12 the metachromatic granules were minute and occurred in only a comparatively small number of the bacilli.

TABLE

Showing the Litmus Broth Reactions of Diphtheroid Bacilli isolated from cases of General Paralysis and Tabes Dorsalis.

No.	Patient.	Disease.	Source of bacillus.	Glucose.	Saccharose.	Starch.	Salicin.	Lactose.	Dextrin.
1	L.	G.P.	Cerebro-spinal fluid	+	–	–	–	–	–
2	M.	G.P. and Tabes	Urine	+	+	–	–	–	...
3	,,	,,	Urine	+	+	–	–	–	...
4	,,	,,	Urine	+	+	–	–	–	–
5	N.	G.P.	Bronchial gland	+	+	–	–	–	...
6	M.	G.P. and Tabes	Urine	+	–	–	–	–	...
7	,,	,,	Urine	+	–	–	–	–	...
8	,,	,,	Urine	+	–	–	–	–	–
9	,,	,,	Urine	–	–	–	–	–	...
10	,,	,,	Urine	–	–	–	–	–	...
11	,,	,,	Urine	+	+	–	–	–	...
12	W.	G.P.	Conjunctiva	–	–	–	–	–	...
13	M.	G.P.	Peritoneum	+	+	–	–	+	...
14	,,	,,	Peritoneum	+	+	–	–	+	...
15	C.	Tabes	Urine	+	+	–	–	–	–
16	G.	Tabes	Urine	+	+	–	–	–	–
17	N.	G.P. and Tabes	Urine	–	–	–	–	–	–
18	G.	G.P.	Cerebro-spinal fluid	+	+	–	–	–	–
19	B.	G.P.	Urine	+	–	–	–	–	–
20	Klebs-Löffler Bacillus (virulent) (from a case of diphtheria)			+	–	–	–	–	–

In this series, among the strains of bacilli having very prominent metachromatic granules, it seems to be possible to recognise two special types, one having the broth reactions of the Klebs-Löffler bacillus, causing an acid reaction in glucose broth only, and the other differing from this in producing acid also in saccharose broth. Two strains, in other respects similar to the latter, were found to produce acid also in lactose broth. It seemed to us at one time that the negative reaction of the first type to dextrin might serve to distinguish it from the Klebs-Löffler bacillus, but we have been unable with the dextrin broths we have used to obtain an acid reaction with the Klebs-Löffler bacillus even after seven days. The reactions given by the bacillus of the second type do not seem to correspond exactly to those of any diphtheroid bacillus hitherto described. The clear differentiation of these two types has been the main result of the application of these tests.

The virulence of six of these strains (Nos. 1, 4, 13, 16, 18, and 19) has been tested upon guinea-pigs. Hypodermic injections of 100 mgm. of agar cultures have produced no apparent effects. With bacillus No. 1 the results of hypodermic injection into mice of doses of from 20 to 60 mgm. have been inconstant. Some of the mice, after showing a few morbid symptoms, remained well. One which received an injection of 40 mgm., and after 23 days another injection of 30 mgm., died three days later. Another mouse injected subcutaneously with 40 mgm. of the killed culture (heated to 128° C. for half an hour) died six weeks afterwards. Another mouse injected with 25 c.c. of immune sheep's serum, taken from a tube containing 5 c.c. of this serum which had been incubated at 37° C. along with 40 mgm. of the bacilli for 48 hours, became acutely ill 10 minutes after the injection, and died within 48 hours. Bacillus No. 4, which, as will presently be described, has caused acute and chronic disease in rats when given in the food, has not proved very virulent to mice when injected subcutaneously. Four successive injections, at intervals of from 9 to 14 days, resulted in the death of one mouse about four weeks after the last injection. Single injections of the usual dose of cultures of bacilli Nos. 8 and 13 had no distinct effect upon mice. Single injections of 40 mgm. of cultures of bacillus No. 18 proved fatal to each of two mice on the fifteenth day.

The Experimental Production of General Paralysis in Rats.

In previous papers we have described the results of experiments carried out by Dr Shennan and one of us, in which rats were fed for several weeks on cultures of a threading diphtheroid bacillus isolated from the bronchus of a general paralytic. It is certain that the organism belonged to the first of the two types that we have reason to believe to be of importance in general paralysis. We have recently made a more complete examination of the tissues of the animals that were the subjects of this experiment, and find that the lesions presented by them resemble those that occur in certain cases of general paralysis even more closely than we have hitherto claimed. All of four animals in which the experiment was fully carried out died after manifesting paretic symptoms, and their cerebral tissues show periarteritis, neuroglia proliferation, and severe nerve cell lesions. Three out

of the four show extensive invasion by the thread form of the bacillus, identical with that which occurred in the patient from whom the bacillus was isolated, and with that which has been found in several cases of general paralysis and in the bladder wall in one case of tabes.

Last autumn we commenced another series of similar experiments with rats. Sixty animals in all have been fed with cultures of various strains of diphtheroid bacilli derived from cases of general paralysis or of tabes dorsalis. These cultures were simply added from time to time to food similar to that upon which control animals were fed. The strains selected were Nos. 1, 4, 8, 13, and 16 in the foregoing list. Very small doses were given during the first three or four weeks, the object being to immunize the animals if possible, and to ascertain the nature of any morbid changes that might occur as the defences of the animals gradually broke down before the persistent attack of the bacilli. This plan was adopted because in the previous series of experiments the rats succumbed far too quickly to permit of advanced chronic changes being established. A certain number of the animals have already died, but the experiments are not yet completed, and we shall therefore here deal only very briefly with some of the results so far obtained.

The first experiment has consisted in the feeding of twelve animals with cultures of a diphtheroid bacillus isolated from the cerebro-spinal fluid of a case of general paralysis post-mortem. It was originally a threading bacillus, and it was the only organism that developed in the media. It produces acid in glucose broth only. It is undoubtedly a bacillus identical with the one used in the previous set of experiments upon rats. As yet only three of the animals have died. They did not exhibit very evident paretic symptoms. All of them were found to have advanced tuberculosis affecting the lungs. The cerebral changes are of too slight a nature to be regarded as of any importance. Some of the remaining rats are now showing signs of paresis. It is evident that this organism is one of much less virulence to rats than the bacillus used in the experiments carried out in 1903.

In the second experiment twelve rats have been fed with a bacillus isolated from the urine of a case of tabo-paralysis. The organism has prominent metachromatic granules, but has never shown any tendency to form threads. It produces acid rapidly

and abundantly in glucose broth, somewhat less rapidly in saccharose broth. This bacillus has proved much more virulent than the preceding one. After about seven weeks, one of the animals was seen to be acutely ill. It was slow in its gait, feeble, and extremely irritable. Next day it was paralysed in all its limbs. It lay for several hours quite motionless, excepting for the respiratory movements, which gradually became reduced in number until they occurred only four times per minute. The animal died within thirty hours of the first appearance of acute symptoms. Tubes inoculated from the heart's blood and spinal cord remained sterile. The chief morbid change revealed by microscopical examination is acute degeneration of most of the nerve cells of the spinal cord. The nerve cells of the brain present similar changes, but they are much less severe. A few days after the death of this animal, two other rats began to show similar but less marked symptoms. After four or five days the acute symptoms passed off, but the animals remained lethargic and feeble. Two months later both of them were noted to be more feeble, very slow and uncertain in their gait, and much emaciated; they were dull, drowsy, and stupid. All of these symptoms gradually increased, and the two animals died within about a fortnight of each other, twenty-two and twenty-four weeks from the commencement of the experiment. The remaining nine rats have never had any acute illness, but all of them have gradually become thin, enfeebled, lethargic, impaired in their power of co-ordination, and stupid-looking. Five of them are now dead. The four that remain are extremely feeble, irresponsive and demented-looking. All of the seven rats of this series that have succumbed to a chronic illness show similar changes affecting the nervous system, differing only in degree in the individual cases. There are extremely well-marked chronic degenerative changes in the nerve cells of the spinal cord; less advanced changes of a similar character in the nerve cells of the cerebral cortex. In several of the cases the cerebral arterioles show distinct periarteritis, and the neuroglia also shows some proliferative changes. Cultures were made from the blood and various internal organs of most of these rats, but no growth of the bacillus with which the animals had been continually fed could be obtained.

In the rats, the subjects of the three other experiments, the

morbid phenomena have been much less distinct, but paretic symptoms are now developing in some of the animals in two of the cages.

On the ground of these and other observations that we shall allude to presently, we maintain that there are at least two different species of diphtheroid bacilli that are capable of producing general paralysis and tabes dorsalis. The one is an organism which morphologically and in its broth reactions, in so far as we have yet been able to ascertain them, resembles the Klebs-Löffler bacillus, but is distinguished from it in frequently being virulent to mice and rats, and in having a thread form which is assumed when it invades the partially immunized animal and under analogous conditions *in vitro*. It is the organism for which we have already suggested the name *bacillus paralyticans*. The other species of diphtheroid bacillus is one which has a very similar relationship to the xerosis bacillus, which by some observers has been found to produce a small amount of acid in glucose and saccharose broths. It differs from it, however, in showing prominent metachromatic granules, in producing acid quickly and abundantly in glucose and saccharose broths, and in frequently being virulent to mice and rats. This organism has little or no tendency to form threads. It is thinner and shorter than the other. We therefore propose to refer to the first organism as the *bacillus paralyticans longus*, and to the second as the *bacillus paralyticans brevis*. Just as it is known that individual strains of the Klebs-Löffler bacillus vary considerably in their microscopical and cultural characters and in their virulence, so also individual strains of these two species that have proved capable of causing paresis in rats present differences that cannot be regarded as essential. Chief among these differences is that of virulence. Corresponding to infection by one or other of these two types of diphtheroid bacilli there are, we maintain, two different types of general paralysis, closely resembling each other and yet essentially distinct. Cases of infection by both organisms are probably common. We believe that in all cases of advancing general paralysis and tabes dorsalis one or other of these bacilli, or some other organism endowed with similar pathogenic powers, is gaining access to the lymph stream and to the blood. It seems to us very probable that we have not yet differentiated all

of the species of diphtheroid bacilli that may occur in such cases and exercise a pathogenic action of a similar nature.

Intra-corpuscular Bacteriolytic Indices of Immunized Sheep and of Persons suffering from General Paralysis and Tabes Dorsalis.

In a previous paper we have described a method of estimating the bacteriolytic power of the polymorpho-nuclear leucocytes in relation to the *bacillus paralyticans*, and have contended that, as a rule, the index expressive of this power is abnormally high in general paralytics. In the course of the immunization of four sheep with bacillus No. 1 in the foregoing list, we have endeavoured to ascertain the effect of the process upon this index. The following table shows the results.

Sheep.	Before Immunization.	Time after commencement of Process of Immunization.		
		Six Weeks.	Six Months.	Thirteen Months.
No. 1	4·3	41	...	73
No. 2	0	48	...	78
No. 3	2·5	...	...	59·5
No. 4	6	...	85	...

These results seem to us to justify the conclusions we have already formed in regard to these indices in cases of general paralysis, although in the human subject the matter is complicated by the circumstance that more than one species of diphtheroid bacillus is capable of causing the disease. The following table shows the observations that we have made upon the human subject during the past year.

No.	Nature of Case.	Bacillus Used.	Index.
173	Control. Healthy	B. p. longus	13
187	Dementia Præcox	,,	17·5
188	Control. Healthy	,,	8
189	,, ,,	,,	16
190	,, ,,	,,	28
196	,, ,,	,,	12·5
170	General Paralysis	,,	76
171	,, ,,	,,	75
172	,, ,,	,,	74
174	,, ,,	,,	92
175	,, ,,	,,	78·5

No.	Nature of Case.	Bacillus Used.	Index.
177	General Paralysis	B. p. longus	83·5
186	,, ,,	,,	67
191	,, ,,	,,	61
192	,, ,,	,,	81
197	Tabes Dorsalis	,,	7·5
		B. p. brevis	61
198	General Paralysis	B. p. longus	44
		B. p. brevis	42·5
199	,, ,,	B. p. longus	66
		B. p. brevis	37
200	,, ,,	B. p. longus	17
		B. p. brevis	71

Evidence of the Rapid Destruction of the Bacilli in the Blood.

Although we have succeeded in obtaining cultures of diphtheroid bacilli from the blood and from the cerebro-spinal fluid of general paralytics during life, there can be no doubt that in the vast majority of cases it is impossible by any method as yet known to obtain such cultures. We have maintained that this difficulty is due, not necessarily to the absence of the bacilli from these fluids, but to the fact that the organisms are quickly taken up by phagocytic cells and devitalised and dissolved. We have made some observations upon sheep in process of immunization to the *bacillus paralyticans longus*, the results of which are consistent with this explanation, as well as with the failures to obtain growths from the blood of general paralytics.

Two sheep, which had previously received seven injections of bacilli in the course of about five weeks, were each injected subcutaneously with 40 mgm. of living bacilli, and two hours afterwards several large platinum loopfuls of blood were smeared upon agar surfaces. The tubes were allowed to remain in the cold for two hours and were then incubated. No diphtheroid bacilli appeared. In blood films made at the same time as the inoculations of the tubes, diphtheroid bacilli could be seen in considerable numbers, especially in the lymphocytes.

After the immunization of these two sheep had been continued for over six months, inoculations were again made from the blood four hours after the subcutaneous injection of 80 mgm.

of bacilli into each. Two agar tubes and one broth tube were used. All of the tubes from the first sheep remained sterile. In the case of the second sheep a few colonies of the bacilli appeared in one of the agar tubes and there was also a growth of the organism in the glucose broth.

Observations in a Case of Tabes.

We also wish to mention some special observations we have made in a case of early tabes dorsalis, as they support in a remarkable way some of the views that we have advanced. The patient's urine was found to be loaded with two micro-organisms, a diphtheroid bacillus of the type described as the *bacillus paralyticans brevis*, and a diplococcus resembling the gonococcus, but Gram-fast. For a time we treated the patient by the vaccine method, injecting definite doses of killed cultures of the bacillus isolated from his urine. On several occasions these injections were succeeded after a few hours by a recurrence of the patient's lightning pains. It was quite clear that these attacks were consequent upon the injections. Very considerable improvement took place in the patient's condition when this treatment had been continued for some weeks. His attacks of pain became much less frequent and less intense. Having with the same bacillus immunized a sheep in such a way as to produce a bactericidal serum, we treated the patient for several weeks with this serum, given first hypodermically and subsequently by the mouth. This treatment has resulted in further improvement, but it has been found that if four or five days are allowed to elapse without a dose of the serum there is still some return of the pains. It is also of interest to note that in this case the urine is now sterile. The patient has been taking helmitol for about six months. In another case of tabes with the same two organisms in the bladder, the continuous administration of helmitol has been followed by the disappearance of the diplococcus, but the bacillus is as abundant as ever. It therefore seems probable that the eradication of the bacillus from the bladder in the first case was due to the action of the serum

The Infective Foci in General Paralysis and Tabes Dorsalis.

In previous papers we have maintained that in all advancing cases of general paralysis and tabes dorsalis there is some more

or less extensive infective focus in which the pathogenic bacilli are invading the tissues. We have obtained histological evidence of the presence of such foci in the alimentary tract and bronchi of general paralytics and in the bladder of tabetics. In the course of the past year we have made some observations upon the living subject which seem to us to confirm the doctrine of the infective focus, and also to throw some fresh light upon the subject.

In association with the presence of abundant diphtheroid bacilli in the female genital tract, we have, in two cases, found scrapings from inflamed cervix to show numerous diphtheroid bacilli in the interstices of the tissues, proving that the bacilli were invading. Similarly, in two cases of chronic conjunctivitis in general paralytics, scrapings from the conjunctiva showed diphtheroid bacilli with prominent metachromatic granules invading the tissues. In another case of advancing general paralysis a scraping from the nasal mucosa, taken from about the middle of the septum, showed abundant similar diphtheroid bacilli lying between the epithelial cells and among the connective tissues. Further, we find that general paralytics usually have more or less swollen and spongy gums, which readily bleed. This morbid condition occurs especially in proximity to the teeth. Between the tooth and such a swollen gum there is almost constantly a distinct pocket in which material collects, and this material, according to our observations, is loaded with diphtheroid bacilli showing prominent metachromatic granules. In fifteen cases of general paralysis presenting this swollen and spongy condition of the gums we have made a microscopical examination of the spongy tissue, after scraping some of it away with a sharp spoon. In thirteen of the cases diphtheroid bacilli have been recognised in the tissue spaces.

We have previously advanced evidence in support of the view that in tabes dorsalis the special infective focus is, in most cases, in the bladder walls, and we have pointed out how this opinion harmonises with the results of the experimental work of Orr and Rows. The more recent observations that we have made in cases of tabes have served to confirm our view, which we maintain to be correct, notwithstanding the criticisms that have passed upon it. We would, however, extend the possible area of the infective focus to the whole of the genito-urinary

tract and the lower portion of the alimentary canal. We now feel justified in drawing a similar inference with regard to general paralysis, and in stating that the infective focus in this disease is chiefly located in the buccal and naso-pharyngeal mucosæ. We assert that the clinical phenomena of amimia, affecting mainly the lower part of the face, dysarthria, and facial tremors are essentially dependent upon this local invasion and consequent formation of toxines, which are in large part carried to the cranial cavity by way of the cranial nerves. This view is also in harmony with the more recent experimental observations of Orr and Rows (10), who have found that, just as tabetic lesions can be produced by toxines passing up the sheaths of the spinal nerves, so also certain of the central lesions that occur in general paralysis can be produced by toxines that are experimentally made to pass up the sheaths of the cranial nerves. On the ground of their observations they definitely apply the lymphogenous theory of infection to general paralysis as to tabes.

There are one or two points that we should like to emphasise in concluding this paper.

The diphtheroid group is a very much more extensive one than has generally been supposed, and the bacillus of acute diphtheria is by no means the only member of the group that is pathogenic to man. Within the last two or three years the multiplicity of species in the diphtheroid group has come to be recognised by various workers, more especially by Gordon in this country, who has directed attention to the fact that many of the organisms that have commonly been regarded as non-virulent diphtheria bacilli give an acid reaction with saccharose broth, and by Hamilton and Horton (11) in America, who have isolated very numerous species. We would endorse the opinion of Benham that the further study of this group will probably lead to great advances in our knowledge of the pathology of some obscure diseases.

In this large group of organisms there are at least two species which are capable of causing paresis in rats, and which can be isolated especially from cases of general paralysis and tabes dorsalis. In patients suffering from these diseases, as well as in other persons, the mere presence of these organisms upon a

mucous surface is of little significance. It is necessary to find evidence of invasion of the tissues by the bacilli. The occurrence of such invasion can be demonstrated in cases of general paralysis. There are good grounds for believing that a bacillus so invading tends gradually to become raised in virulence, especially towards the individual attacked. The rational therapeutic aim in cases of general paralysis and of tabes dorsalis ought to be to eradicate the infective focus. Our own researches during the past year have been mainly directed to this end, and we have used as our chief therapeutic agents bactericidal sera corresponding to the two varieties of diphtheroid bacilli we have found to be capable of producing paresis in rats. The work has been beset with very many difficulties, chief among which, we are now realizing, have been those occasioned by loss of virulence on the part of the organisms we have been using. Notwithstanding this, the results of serum treatment have in many instances been very encouraging.

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Abstracts

ANATOMY.

THE COMPARATIVE ANATOMY OF THE CEREBELLUM.

(226) L. EDINGER, *Brain*, 1906, p. 482.

IN this preliminary note Edinger shortly summarises his observations on the comparative anatomy of the cerebellum.

1. The size of the cerebellum varies directly with the equilibratory needs of the animal; *e.g.* it is small in the mud-living fishes and land reptiles, large in the swimming fishes and water reptiles.

2. In all animals the cortex and lateral intrinsic nuclei are differentiated. The latter are probably homologues of the nucleus dentatus, as the superior cerebellar peduncles spring from them. The median (fastigial) nuclei appear first in the birds and mammals, but the hemispheres only in mammals.

3. In the lower vertebrates the afferent and efferent tracts pass through a single peduncle.

(A) The *afferent tracts*, which originate in the primary end-stations of the various afferent nerves, come from—

(1) The cord from Clarke's column by the dorsal and ventral spino-cerebellar tracts.
(2) The dorsal column nuclei.
(3) From at least the 5th, 8th, and 10th cranial nerves associated with certain direct root fibres of these nerves.
(4) From the primary optic centres in fishes, amphibians, birds, and reptiles.

These afferent tracts run in the most caudal portion of the peduncle, and cross to the contralateral side of the cerebellum.

(B) *Efferent tracts.* In all vertebrates the cerebellum sends out a large tract to the tegmentum. It probably originates in the intrinsic cerebellar nuclei. It is divisible into a bulbar portion to the nucleus lateralis medullæ region, into a spinal portion which also contains the vestibulo-spinal fibres, a tectal portion to the tegmentum of the mid-brain, and finally fibres to the hypothalamus. The latter two divisions constitute the superior peduncles in the higher mammals. GORDON HOLMES

A CASE OF CROSSED ATROPHY OF THE CEREBELLUM. ANDRÉ-(227) THOMAS et R. CORNELIUS, *Rev. Neurol.*, March 15, 1907.

THE brain described was from a woman who died at the age of 61. From the age of 10 years she had been an epileptic; and at 41, that is, twenty years before her death, she developed left hemiplegia with hemianæsthesia after a fit.

The right hemisphere of the brain was found very much atrophied, especially the frontal lobe; the Rolandic region and the occipital lobe were the least affected. The sagittal layer of the temporo-occipital lobes, the tapetum, and the corpus callosum were also atrophied, but Türck's bundle escaped. The corpus striatum was unaffected; but the anterior, median, and lateral nuclei of the thalamus were much wasted, as well as the pulvinar and Wernicke's field. The inner $\frac{4}{5}$ of the crus was reduced in size, but the outer $\frac{1}{5}$ containing Türck's bundle was normal. The pons was asymmetrical, the right side being smaller than the left; this was due to atrophy of the pontine grey matter, the transverse fibres being unaffected. There was marked atrophy of the left lateral lobe of the cerebellum, greatest in the quadrate lobe, less, but still distinct, in the superior and inferior semilunar, the gracilis, and the digastric lobes. The flocculus was not affected, but the central nuclei of the cerebellum were diminished in size on the left side. On microscopical examination it was seen that the Purkinje cells had disappeared from parts of the atrophied lobe; none could be found in the left third of the superior vermis, but they persisted in natural number in the inferior vermis. The three cerebellar peduncles of the left side were definitely atrophied; and, as a result of the atrophy of the left corpus restiforme, the right inferior olive was considerably wasted and sclerosed, especially its superior half, in which very few nerve cells could be found. The several accessory olives appeared unaffected.

As in the majority of the cases which have been described, so in this case the hemiatrophy of the cerebellum was associated with a diffuse and not with a focal lesion of the opposite cerebral hemisphere. Although the hemiplegia supervened only at the age of 41, the affection of the right cerebral hemisphere probably dated from much earlier in life; microgyria, which was a marked feature of this hemisphere, occurs only with congenital lesions or lesions which date from infancy, and it is only with such lesions that atrophy of the opposite cerebellar hemisphere is found associated. In some of these cases the hemiatrophy of the cerebellum has been attributed to lesions of the cerebral cortex and the resulting degeneration of the pontine nuclei and the opposite middle peduncle, in other cases to thalamic lesions and the consequent atrophy of the superior peduncle; but the authors have come to

the conclusion that it is not possible to attribute the hemiatrophy of the cerebellum in their case exclusively to either a cortical or a thalamic lesion.

The wasted cerebellar lobe had on the whole the histological characters of an agnesia rather than of an atrophy, but the disappearance of the Purkinje cells indicated a regressive or degenerative change. GORDON HOLMES.

(228) **NEW STUDIES ON THE FOLDING OF THE VISUAL CORTEX AND THE SIGNIFICANCE OF THE OCCIPITAL SULCI IN THE HUMAN BRAIN.** G. ELLIOT SMITH, *Journ. Anat. and Phys.*, vol. xli., April 1907.

THE paper is itself an abstract of a communication read before the Anatomical Society at Belfast in June 1906.

If a coronal section be made through the occipital region (about 1 cm. behind the fossa parieto-occipitalis) in a *perfectly fresh* human cerebral hemisphere, one can recognise (in most cases), with the unaided eye, eight strips of cortex, each differing from adjoining areas in various respects. For reasons given the naked-eye examination of fresh specimens is preferable to the examination of sections stained by Weigert's method, or of reconstructions from sections stained by Nissl's method.

The anatomical localisation of three areas—the area striata, the area parastriata, and the area peristriata—and the causal relationship of their distribution to a large series of sulci is considered and illustrated by diagrams.

The relationship of the area striata, *i.e.* the cortex containing the stria of Gennari, to various fissures, including the sulci cunei et lingualis and the anterior calcarine fissure, is described; and distinctions are drawn amongst three different varieties of sulci related to the area: (1) *Sulci limitantes*, in which the area striata extends to the bottom of the furrow, forming one of its walls as in the sulci above named; (2) *Sulci operculati*, where the area reaches the lip of the sulcus as in the case of the polar sulci of Bolton and the sulcus lunatus; and (3) *Axial furrows*—the posterior and the external calcarine—where the area has been folded and forms both walls of the furrow.

The area parastriata is distinguished by the absence of a definite line of Baillarger, and the layer of grey matter between the medullary white centre and the pale inner half of the cortex is but faintly marked. In the area peristriata, which lies beyond the ring of area parastriata, the diffuse paleness of the inner half of the cortex gives place to a definite band of Baillarger, and a definite grey layer separates the band from the medullary centre.

The limits and the furrows in relation to these areas are described.

From the area peristriata a process stretches forwards between the parietal and temporal association areas of Flechsig, and is identified as "area 12" (gyrus subangularis) of Flechsig.

E. B. JAMIESON.

ON THE MODE OF TERMINATION OF THE CEREBRAL
(229) **ARTERIES. (Du mode de terminaison des artères cérébrales.)** LOUTEN, *L'Écho Médical du Nord*, Jan. 6, 1907.

THE writer has made a study as to the communications between arterial branches in the brain by means of injections of red wax and soluble Prussian blue. His results are as follows :—

There are on the surface of the brain three territories corresponding to the distribution of the anterior middle and posterior cerebral arteries, but these encroach upon one another, communication being effected through the small arteries of about one half millimetre in diameter. The arteries of the two hemispheres sometimes communicate through a mesial artery of the corpus callosum; but the writer entirely failed to find any of the so-called canals of Sucquet leading directly between arteries and veins. The arteries are not, as is generally supposed, terminal from the pia mater inwards, but they give off branches which form a meshwork in the substance of the brain from which the terminal nourishing arterioles are given off. Some of the latter form their arborisations in the grey matter, other longer ones form theirs in the white matter, and the systems of neighbouring arterioles again anastomose, although to a very limited extent. The writer denies, however, that any communication exists between the central and the superficial systems of arteries, although the branches of the latter penetrate the white matter to a depth of 4 or 5 centimetres.

A full bibliography of the subject is given.

JOHN D. COMRIE.

EXPERIMENTAL RESEARCHES ON THE MORPHOLOGY OF
(230) **CELLS AND FIBRES OF THE SPINAL GANGLIA. (Recherches expérimentales sur la morphologie des cellules et des fibres des ganglions rachidiens.)** NAGEOTTE, *Rev. Neurol.*, No. 8, 1907, p. 357.

SOME ganglion cells have various kinds of prolongations, others fenestration of the axis-cylinder and protoplasm, and others are surrounded by pericellular or periglomerular masses of fibres.

The rôle of these is obscure, but the author has already noted the presence of processes, terminating in bulbs, in tabes, and has ascribed some regenerative function to them.

In these experiments the ganglia were transplanted, and the author draws attention to three special new formations. *Nodular arborisations.*—In the situation of the degenerated nerve cells there are nodules formed by the persistence of subcapsular cells (satellites). These are rich in protoplasm, and their nucleus is peripheral in situation. Branches from the axis-cylinders of neighbouring cells penetrate these nodules and ramify there, especially in their central part. *Periglomerular arborisations.*—The fibres forming these are arranged in irregular spirals around the glomeruli of surviving cells. After complete absorption of the destroyed nerve cells, the ramifying fibres in question continue to grow and form the nodules mentioned above and the pericellular nests to be described. It would appear as if some substance was secreted at this stage which attracted the axis-cylinders towards the satellite cells. *Pericellular nests.*—These exist normally, but are relatively increased in numbers in tabes. In the transplanted ganglia they are present in considerable numbers, and the fibres forming them come from the axis-cylinder of the cell itself, around which the mass of fibres forms.

The fibres forming all three structures are supposed by the author to be drawn from axis-cylinders by the chemiotaxis of the satellite cells.

In spinal and sympathetic ganglia there are some nerve processes which are neither in contact with other cells nor with muscles or sensory organs. Nageotte calls these paraphytes, in contradistinction to orthophytes, which form connections with other neurons. Some paraphytes possess a tropism; others do not normally, but acquire it and multiply whenever nerve repair is necessary. DAVID ORR.

PHYSIOLOGY.

(231) THE GROUPING OF AFFERENT IMPULSES WITHIN THE SPINAL CORD. By HENRY HEAD and THEODORE THOMPSON, *Brain*, 1906, pp. 537-741.

THIS long communication is a second instalment of an epoch-making research in the field of sensation. Any abstract of it can give but an imperfect idea of the immense amount of material, methodically and minutely handled, on which the argument is based. For this alone the paper demands study *in extenso*. After reading and re-reading it, one feels that our clinical examination

of cases from the sensory point of view has hitherto been lamentably incomplete, and that no one can afford hereafter to come short of the standard set before themselves by the authors.

From an examination of the phenomena which follow division of peripheral nerves, it has been found that the afferent mechanism at this level of the nervous system consists of three systems:—

(1) A system corresponding to the group of deep sensibility impulses; that is, the sense of pressure (painful or otherwise), and of the movement of joints, tendons, and muscles. The fibres which conduct these sensory impulses run mainly with the muscular nerves, and are not destroyed by division of all the sensory nerves to the skin.

(2) The protopathic system, capable of responding to painful cutaneous stimuli and to the more extreme degrees of heat and cold.

(3) The epicritic system, by which we localise points touched on the skin, discriminate two points, and recognise the finer grades of temperature, called cool and warm.

Now, on comparing the loss of sensation produced by disease and injury of the spinal cord with that caused by division of peripheral nerves, fundamental differences at once become apparent; and these it is the object of this paper to elucidate.

The chapter on methods must be read *in toto*, as it is replete with suggestions of practical value, and contains an exposure of numerous fallacies which the authors have met with in their experience. Emphasis is laid on the importance of recording in a definite manner the results of examination. It is suggested, for instance, that in testing with compass points for the power of discrimination, the patient should be touched ten times with one point, ten times with two points. Each series is recorded in such a manner that it becomes immediately obvious in how many cases among the ten single and among the ten double stimuli the patient has given a wrong answer. Similarly, in regard to passive movements, ten are made in one direction, ten in the other, indiscriminately, and the results are marked above and below a line, each answer being recorded by a stroke if right, by a cross if incorrect. If no answer is given, an 0 is inserted, whilst doubt is indicated by a D. Such a method greatly increases the accuracy of the record. A modification of Cattell's algometer, of which a full description is given, was used for testing sensations of pressure. In testing the certainty with which a patient can indicate the point touched, (1) he was asked to name it; (2) he was told to point to the spot stimulated; (3) a hole was cut in the centre of a stiff sheet of paper, and through this he was stimulated, the paper being held about 1 cm. distant from the skin. He was given a soft pencil, and with this tried to point to the spot stimulated.

The point he reached was marked on the paper, and the distance from the central hole could be measured. (Spearman's method.)

Remembering the three systems of the afferent mechanism at the primary (peripheral) level, we may make the following summary:—

Loss of *epicritic sensibility* abolishes—

Recognition of light touch.
Cutaneous localisation.
Discrimination of compass points.
Appreciation of differences in size.
Discrimination of intermediate (25° C. to 40° C.) degrees of temperature.

Loss of *protopathic sensibility* abolishes—

Cutaneous pain (pricking, burning, freezing, electric, hair-pulling).
Sensations of heat above 40° C., and of cold below 20° C.

After destruction of all cutaneous afferent fibres the part is still endowed with *deep sensibility*.

Pressure is recognised and its increase appreciated.
Pain is produced by excessive pressure.
Movements of muscles can be recognised.
The point of application of pressure can be recognised.
The extent and direction of passive movement can be recognised.

Now, if the lesion is within the spinal cord, notable differences occur.

1. If sensibility to pain is abolished, *all* forms of painful stimuli are simultaneously affected.

2. Sensibility to heat may be abolished with coincident disturbance of that to cold, and *vice versa*. When sensibility to heat is disturbed in consequence of an intramedullary lesion, the patient no longer appreciates any thermal stimulus between 30° C. and 60° C. That is to say, insensibility is absolute to both intermediate and extreme degrees. Insensibility to heat and cold may be absolute, and yet the patient may be able to recognise the lightest tactile stimulation, and to discriminate the two points of the compasses.

3. If sensibility to touch is abolished in consequence of an intramedullary lesion, all forms of tactile stimuli will be found to be affected. The peripheral afferent impulses for touch and pressure, arriving by way of the epicritic and deep systems, become combined in the spinal cord.

4. In intramedullary lesions, every other form of sensibility may be abolished in a part which still remains sensitive to passive

movement. Or the patient may be unable to recognise even the grossest passive movements, although sensitive to all tactile and pressure stimuli. Or the sense of passive position and movement may be disturbed in one leg, while every other loss of sensation may be found in the leg of the opposite side.

5. Within the spinal cord the impulses which underlie the power of discriminating two points are separated from those of tactile sensibility.

For the purpose of elucidating the *inter-relation of afferent impulses in their passage up the spinal cord*, eight cases of Brown-Séquard paralysis observed by the authors are relied on.

A. *Disturbances of Sensation remote from the Level of the Lesion.*

1. Afferent impulses from pain, heat, and cold cross the spinal cord to pass up the opposite side. The remote loss of sensation may affect to a varying degree the impulses for the three forms of sensibility. The borders of this remote loss of sensation may correspond to the borders of intramedullary segments: sometimes they merge gradually into parts of normal sensibility.

2. In Brown-Séquard paralysis, if disturbance of sensibility to touch and pressure occur, it is found on the opposite side of the body to the motor disturbance. The impulses of light touch, deep touch, and the tactile element of pressure are lost together.

3. The sense of passive position and movement is abolished on the same side as the motor disturbance. It is independent of the tactile impulses and of those which underlie the power of cutaneous localisation.

4. If all forms of tactile sensibility are perfect in Brown-Séquard paralysis, the power of discriminating two points will be found to be diminished over the side of the motor disturbance. There is a close relation between loss of the sense of passive position and movement and this power of discriminating two points; loss of the one is usually associated with grave disturbance of the other.

B. *Local Effects of an Intramedullary Lesion.*

A fundamental difference between the local and remote effects of intramedullary disease is that every disturbance of cutaneous sensibility due to the local effect of disease of the spinal cord lies on the same side as the muscular wasting and loss of motor power, that is, on the side of the maximum lesion. On the other hand—

1. The local action of a lesion within the spinal cord may produce a more extreme thermo-anæsthesia to heat than to cold, or *vice versa*.

2. The analgesia produced by the local manifestations of an intraspinal lesion consists essentially of insensibility to the pain of both cutaneous and deep stimulation, and corresponds exactly with that due to its remote effects.

3. Sensations of light touch and the tactile aspect of pressure are linked together.

4. When the loss of sensation at the level of an intraspinal lesion includes disturbance of the sense of passive position and movement, the discrimination of two points is also affected.

The Passage through the Spinal Cord of Afferent Impulses concerned with "Localisation."

The sense of passive position and movement may be destroyed as a consequence of intramedullary disease, although sensibility to deep touch remains perfect. Conversely, the patient may be insensitive to all tactile stimulation, both superficial and deep, and yet may be normally sensitive to passive movements. Any loss of the sense of passive position and movement will be found on the same side of the body as the disturbance of motion and reflexes. Further, the power of discriminating two points can be diminished over a limb completely sensitive to all cutaneous stimuli. If, as a consequence of the remote effects of an intramedullary lesion, the compass test shows a diminution of sensibility whilst touch is perfectly appreciated, the part so affected will lie on the same side as the disturbance of motion. Again, the impulses underlying spot-naming run independently of those for tactile discrimination and the recognition of passive position and movement.

The impulses underlying the latter two differ from every other sensory impulse passing up the spinal cord, since they do not cross until the medulla and pons are reached. In the cord they have not reached the second level of the sensory nervous system. For these impulses we must seek some path in the spinal cord which is the direct continuation of the posterior roots, and which remains uncrossed until we have passed beyond the limits of the spinal cord. Such a path is found in the long extrinsic tracts of the posterior columns.

Painful, thermal, and tactile impulses ultimately pass from the point of entry to the opposite side of the spinal cord. Before they pass across they undergo characteristic recombination. That this change occurs in the same half of the spinal cord by which the impulses enter, is shown by the fact that the local loss of sensation produced by a pure intramedullary lesion closely resembles, in character and grouping, that of the remote sensory disturbance which lies over the opposite half of the body. The rapidity with which these sensory impulses cross to the opposite side varies greatly.

It is probable that the fibres of the deep system, carrying impulses produced by painful pressure from the same part of the body, do not enter by the same posterior roots as those carrying the impulses produced by cutaneous painful stimuli. Thus, more than one segment of the cord is required before all the painful impulses from any one part of the body can be gathered together and recombined. Hence a certain extent of the spinal cord must be traversed before the transformed impulses from any part of the body can be gathered up in a path on the opposite side of the spinal cord. Interruption at any point after they have crossed may produce a remote analgesia with a definite upper border, but this border will probably lie several segments below the position of the interrupting lesion.

The delay in crossing and recombination of the impulses subserving the sense of passive position and movement is in part, at least, due to the fact that certain of these afferent impulses never reach consciousness: they may be associated with some higher reflex condition, such as the maintenance of equilibrium or the tone of muscles. As the cerebellum is the afferent centre for these higher reflexes, any column which conducts impulses from the tendons and joints will probably give off collaterals to afferent cerebellar tracts.

The mechanisms of the secondary or intramedullary level are concerned with the separation of non-sensory from sensory afferent impulses, and with the recombination and transmutation of sensory impulses into specific groups. S. A. K. WILSON.

ON THE PROPRIOCEPTIVE SYSTEM, ESPECIALLY IN ITS
(232) **REFLEX ASPECT.** SHERRINGTON, *Brain*, Part cxvi., 1906, p. 467.

THE term "receptor" is applied by the author to the peripheral apparatus which, excited by an adequate stimulus, reacts so as to cause the initiatory impulse in a natural reflex. It is a matter of secondary importance whether the reaction of such a receptor is attended by sensation.

The main fields of distribution of the receptor organs appear to be two, namely, a surface field constituted by the surface layer of the organism, and a deep field constituted by the tissues beneath the surface sheet.

The surface field is subdivisible into a truly cutaneous or exteroceptive surface, and an interoceptive surface turned inwards on the alimentary contents.

While the receptors of the exteroceptive field show great variety of adaptation to the different stimuli, those of the interoceptive field are less known.

The receptive field lining the digestive chamber appears especially adapted to chemical stimuli, but the interoceptive system appears from the number of fibres in the sympathetic system to be relatively sparsely endowed with receptor organs. Further, as shown by the work of Bayliss and Starling on the pancreas, excitation of an organ may take place without intervention of the nervous mechanism. The character of the stimulations occurring in the deep field are in the main traceable to the actions of the organism itself. Such deep receptors may be termed proprioceptors. The reactions due to the stimulation of the organ by itself are, however, secondarily due to stimulation of the organism by its environment.

It would thus appear that in many cases the proprioceptive reflex which ensues as a secondary reaction allies itself in its effect to the primary reflex excited by the extero-receptive surface and reinforces it. The flexion reflex of the hind limb of a dog can be excited both by a cutaneous stimulus and by stimulation of the afferent nerves of the flexor muscles themselves. A subliminal stimulation of the skin of the foot becomes effective when a subliminal stimulation of one of the flexor muscles is delivered synchronously. Similarly a flexion reflex of the limb provoked from the skin can be cut off by reflex inhibition on stimulating the afferent nerve of the flexor muscle of the opposite limb. In this case the proprioceptors evoke a compensatory reflex in the opposite direction.

A third character of the proprioceptive field appears to be its tendency to induce and maintain tonic reactions in the skeletal musculature.

The tonic extensor rigidity in decerebrate animals appears to be mainly due to proprioceptor impulses from the deep structures of the limbs, since section of the main skin nerves does not impair it though tonicity vanishes after section of all the posterior roots.

The reflex tonus may be considered as the expression of a neural discharge concerned with the maintenance of attitude. As the attitude of an animal is a complex built up of a series of segmental postures, the proprioceptors or their reflex arcs have in their sum total to be treated as a proprioceptive system. Further, inasmuch as for purposes of locomotion the most anterior portion of this system will be the most important, so a likeness is traced between the labyrinth and proprioceptor mechanism, the likeness being enforced by the influence of the labyrinth on reflex tonus. Just as the proprioceptors of a limb are in a large measure responsible for the reflex posture and compensatory reflexes of the limb, so are the labyrinthine proprioceptors largely responsible for the reflex posture and compensatory reflexes of the head. The proprioceptors of the limbs give to them a definite reflex attitude,

less in reference to the external world than in reference to other segments of the body. The proprioceptive organ of the head gives to the animal a definite reflex attitude in relation to the external world.

Inasmuch as the cerebellum represents the central receptor organ for labyrinthine sensations, so it would be permissible to describe the cerebellum as the head ganglion of the proprioceptive system. F. GOLLA.

THE FUNCTIONS OF THE ROLANDIC CORTEX IN MONKEYS.

(233) W. A. JOLLY and SUTHERLAND SIMPSON, *Proc. Roy. Soc. Edin.*, Vol. xxvii., 1907.

THE method of electrical excitation was first applied to the brain of the monkey by Ferrier, who employed the faradic current as preferable for accurate work to the galvanic. By means of the latter form of stimulus, Fritsch and Hitzig had previously demonstrated that an area of the cortex in the neighbourhood of the crucial sulcus could, in the dog's brain, be mapped out from the rest of the cortex. Excitation of this area gives rise to definite muscular movements.

According to Ferrier the motor area in the monkey comprises the ascending frontal and parietal gyri and part of the angular gyrus. Within this area he represented the centres as roughly circular in outline. The limits of the area ascertained to be excitable were extended by the researches of Horsley and Schäfer, who established the fact that it extends to the mesial face of the hemisphere. By the use of interrupted induction shocks with the coil fitted with Helmholtz's side wire, these observers showed that minimal stimuli elicited from part of the marginal gyrus contraction of perfectly definite groups of muscles, or in some cases of single muscles, produced more or less co-ordinated movements.

Beevor and Horsley investigated the brain of the anthropoid. They found the upper third of the ascending parietal convolution and the part of the paracentral lobule behind a continuation over the margin of the upper end of the Rolandic fissure to be entirely or almost entirely inexcitable. The lower portion of the ascending parietal convolution they considered to form part of the motor area.

Grünbaum and Sherrington, from a study of the brains of various anthropoids by the method of unipolar faradisation with minimal stimuli, came to the conclusion that the motor area lies wholly in front of the fissure of Rolando. In no case, they concluded, does the motor area extend into the ascending parietal convolution.

In view of Grünbaum and Sherrington's conclusions it became

necessary to determine whether the movements obtainable in monkeys by stimulation of the ascending parietal and other convolutions warrant the inclusion of these in the motor area, or whether the movements are to be explained as due to a spread of current to the adjacent excitable cortex.

The method employed was as follows: After a preliminary investigation of the exposed Rolandic cortex, the minimal strength of stimulus was determined which produced muscular movements on application of the electrode to either side of the fissure of Rolando. A thin plate of vulcanite, one edge of which had been sharpened, was then inserted into the cortical substance at the bottom of the fissure of Rolando to a depth which would permit of its dividing the grey matter without penetrating the underlying white substance. Stimulation of the ascending parietal convolution insulated in this way from the ascending frontal failed to give rise to muscular movements. When the insulating plate was withdrawn, these were again obtained. The same method was applied to the determination of the boundaries of the centres in front of the Rolandic fissure.

Conclusions.—The ascending parietal convolution is non-motor. A centre for foot and digits is found in the marginal gyrus. On the convexity of the hemisphere, close to the margin, lies a centre for tail, perineum, and trunk movements. Within the ascending frontal convolution the representation from above downwards is as follows: Foot, knee, hip, body, shoulder, elbow, wrist, digits, neck, eyelids, upper lip, larynx, opening of mouth, closure of mouth, tongue, and mastication. In front of and within the angle of the precentral gyrus lies a centre for the head and eyes. Above this centre is situated an area from which movements of the head can be elicited. AUTHOR'S ABSTRACT.

PATHOLOGY.

ON THE HYPERTROPHY OF THE HYPOPHYSIS IN ANIMALS DEPRIVED OF THEIR THYROID GLANDS.

(234) **(Sulla ipertrofia dell' ipofisi cerebrale negli animali stirordati.)** A. CIMORONI, *Lo Sperimentale*, Fasc. 1, Ann. 61, 1907.

THE author comes to the following conclusions:—

Hypertrophy of the hypophysis after experimental ablation of the thyroid mechanism is due to the ablation of the thyroid gland itself, and not to ablation of the parathyroids.

The histological appearances in such hypertrophy of the hypophysis have a specific character from the presence of certain cells remarkable for their large size, and the condition may be thus

differentiated from the hypertrophy following on castration. This specific cell proliferation would appear to be due to an augmented functional activity of certain cells which are not to be microscopically differentiated in the normal hypophysis or in the condition of hypertrophy following castration, but which become evident after removal of the thyroid.

A consideration of our recent knowledge with regard to the evidently different function of the parathyroids and the thyroid proper, the former alone being indispensable to life, would make the author's conclusions that hypophysis hypertrophy is not an indiscriminate response to ablation of the whole of the thyroid mechanism appear probable. T. GOLLA.

SOME RESEARCHES ON THE TRANSPLANTATION OF NERVE
(235) **GANGLIA. (Quelques recherches sur la transplantation des ganglions nerveux.)** G. MARINESCO, *Rev. Neurol.*, No. 6, 1907, p. 241.

AUTO-TRANSPLANTATION of plexiform and spinal ganglia was performed on dogs, and the cells examined by Nissl's and Cajal's methods. The ganglia were placed under the skin or alongside the sciatic nerve. The cells rapidly degenerated, and were invaded by polymorphonuclear leucocytes. The satellite cells normally situated around the ganglion cells hypertrophy and proliferate, new vessels are formed which penetrate the ganglion, many of whose cells become multipolar in appearance. Where ganglia were placed alongside the sciatic nerve, many of its fibres were directed towards the cells, and surrounded them with fibres of new formation. According to the author this is the result of chemiotaxis. A certain number of cells situated at the periphery of the ganglion showed signs of repair, and, up to a certain point, new formation. Those which survived the operation sometimes showed abnormal expansions ending in huge swellings; even these cells finally disappeared. The satellite cells persist.

DAVID ORR.

A RARE TUMOUR OF THE SPINAL PIA MATER (CHROMATO-
(236) **PHOROMA).** [**Über eine seltene Rückenmarkshautgeschwulst (Chromatophorom).**] Dr ESSER (of Bonn), *Deut. Ztschr. f. Nervenheilk.*, 1907, Bd. 32, H. 2-3, S. 118.

THIS tumour occurred in a man of 32. There was nothing remarkable about the symptoms; they were typically those of a tumour pressing on the cord in the upper dorsal region. He was operated on, meningitis ensued, and the case came to *sectio.*

The tumour is described as being about $4 \times 1\frac{1}{2}$ cm., lying in the pia mater at the level of the first and second dorsal vertebræ, of soft consistence, and lobulated in character. Microscopically it consisted of spindle-shaped cells with large processes and filled with brown-black pigment granules. The cells are typical chromatophores, and the tumour therefore is what Ribbert calls a chromatophoroma, as being a more correct name than the usual one of melanotic sarcoma.

No other organs were involved. The tumour must be regarded as primary, and apparently only three other primary melanotic tumours of the brain or cord meninges have been recorded.

J. H. HARVEY PIRIE.

CLINICAL NEUROLOGY.

PERIPHERAL AND CENTRAL NEURO-FIBROMATOSIS. J. ROUX, (237) *Rev. Neurol.*, April 15, 1907.

THE record of a case of typical peripheral neuro-fibromatosis with a central lesion probably of the same nature. The patient was a girl, aged 12 years, who for about a year had suffered from fits, occurring every fortnight, consisting of convulsive movements of the left side of the body, and followed by weakness of the left side and diminished vision of the left eye. Examination showed paresis of the left side of the body, with spasticity and marked atrophy of all the tissues of the left arm and leg and asymmetry of the face. In the right occipital region there was a large hemispherical bony protuberance. The skin showed pigmentation and other stigmata of Recklinghausen's disease. The writer considers that there is a central lesion similar to the peripheral one, but is in doubt as to the exact localisation. Several previously recorded cases of a like nature are referred to. HENRY J. DUNBAR.

THE CLINICAL HISTORY AND RECOGNITION OF TUBERCULOUS MENINGITIS. HENRY KOPLIK, (238) *Journ. of the Am. Med. Assoc.*, April 6, Vol. xlviii., p. 1149.

THE author has studied fifty-two cases in the past six years and gives here some of his results. The onset was insidious in nearly all cases. A history of sudden onset is generally erroneous, owing to faulty observation. Rigidity of the neck or spine was absent throughout in 27 per cent. of the cases, and never appeared in the first week. Another distinction from epidemic cerebro-spinal meningitis lies in the fact that hyperæsthesia was absent throughout in 90 per cent. of the present cases. Kernig's sign was present

in twenty-two cases. Koplik considers that the occurrence of the Babinski sign in children over two points to the tubercular nature of the meningitis. A low temperature in a case of meningitis points strongly to tubercle; tuberculous meningitis causes pyrexia chiefly towards the end. Evanescent pareses late in the course of the disease are characteristic. In no case was the leucocyte count above 29,000, and in thirty-five of the cases it was below 20,000. In 66 per cent. of the cases there were fundus changes at the outset. Choroidal tubercle was found in nine out of forty-six cases examined on the second day of the active period of the disease. Koplik attaches great value to obtaining a tympanitic note by Macewen's method of skull percussion, and considers it diagnostic of hydrocephalus—acute or chronic. Lumbar puncture is of the greatest value in fixing the diagnosis beyond doubt, and stress is laid on the fact that the tubercle bacilli can be found in nearly all cases after centrifugalisation, provided sufficient patience be displayed. A predominance of lymphocytes in the fluid is strong evidence in favour of the tubercular nature of the meningitis. Polynucleosis occurs in this disease when great invasion of the bacilli into the fluid takes place, or when a secondary infection is present. The Fehling reducing substance is absent in tuberculous meningitis and the albumen increased. ERNEST JONES.

MENINGISM AS DISTINGUISHED FROM MENINGITIS. FROM (239) AN OTOLOGIC VIEWPOINT. CHEVALIER JACKSON, *Journ. of the Am. Med. Assoc.*, March 30, Vol. xlviii., p. 1078.

THIS paper is based on a study of sixty-two cases in which the meningitic syndrome was present without intracranial inflammation. Thirteen of them are fully reported, and of these three were examined post-mortem. The author appends a series of conclusions, the following of which may be quoted: Without lesion of the meninges there may be a syndrome comprising many of the diagnostic signs of meningitis; before recovery, such cases are often indistinguishable from meningitis. According to etiology these cases may be classified as either reflex, toxæmic, or irritative; otologists are concerned chiefly with the latter two groups. Any meningitic symptom may occur, but the erethistic are much more frequent than the depressive; the full development of pressure symptoms or paralysis will usually exclude meningism, and the readiness with which the symptoms of meningism may be quieted by small doses of morphin is a valuable diagnostic point. These cases are distinct from Quincke's serous meningitis, in that there is no serous trouble. In the course of middle-ear disease the symptoms of meningitis often demand radical operation for cure, even if the mastoid be yet uninvolved.

The author states that demonstrable pathologic changes are absent in the first two groups of cases, but in the irritative cases a non-infective hyperæmia may be found, without serous exudation or leucocytic infiltration. Amongst the symptoms, Kernig's sign was present in only four of his cases; no paralyses were noted in any case. Lumbar puncture, so Jackson says, is of little value, because positive findings only occur late, when other signs of meningitis are obvious. ERNEST JONES.

ON THE RELATIONSHIP BETWEEN SIMPLE POSTERIOR (240) BASIC MENINGITIS AND CEREBRO-SPINAL FEVER.
LANGMEAD, *Practitioner*, April 1907.

GENERAL opinion favours the belief that posterior basic meningitis is sporadic cerebro-spinal fever in a somewhat modified form. Clinically there are many points of similarity between the two diseases. The occurrence of rashes in cerebro-spinal fever—giving rise to the synonym spotted fever—is not nearly so common as generally supposed, and many epidemics have been recorded in which there was no rash. Moreover, rashes do occasionally occur in the posterior basic meningitis of infants. Optic neuritis is common in the fever and amaurosis rare; the reverse is noted in posterior basic meningitis. The writer records ten cases of posterior basic meningitis in children over three and a half years in support of the assumption that age has something to do with the differences in the clinical manifestations. Only one of the ten was blind; five had optic neuritis. Two of the ten had herpes. Eight exhibited a swinging, irregular, pyæmic temperature, a symptom rare in posterior basic meningitis in infants and common in cerebro-spinal fever. All of them yielded an organism in most cases indistinguishable from that of Still. It has been objected that posterior basic meningitis does not occur in epidemics, but recent observations go to show that the cases are not so isolated as was formerly believed. The chief pathological distinction between the two diseases lies in the meningeal area affected; but, on the one hand, in a considerable number of cases of posterior basic there is exudate on the vertex and in the ventricles, and, on the other, in cerebro-spinal fever the posterior base bears the brunt of the infection. The chief bacteriological distinctions are the different appearances of agar cultures, the easier growth of Still's organism in broth, and the fact that the organism of Still could be kept alive for long periods on agar, while that of Weichselbaum quickly died. Recent work, however, proves that the last two of these cultural differences no longer hold good, and Still himself stated they were "the result of natural variation,

and represented rather a modification of characteristics than a distinction in kind." The paper concludes with the record of a case of an infant aged eleven months, which was clinically one of the most virulent forms of epidemic cerebro-spinal fever, but bacteriologically was in every way indistinguishable from posterior basic meningitis. HENRY J. DUNBAR.

EXPERIMENTAL CEREBRO-SPINAL MENINGITIS IN MONKEYS.

(241) SIMON FLEXNER, *Journ. of Experimental Medicine*, Vol. ix., p. 142.

THESE experiments were undertaken with a view to reproducing in monkeys the symptoms and cord lesions of cerebro-spinal meningitis in man.

Cultures which were found by intra-peritoneal injection to be fatal to guinea-pigs were introduced into the spinal canal of monkeys by means of lumbar puncture.

The injection was made at the level of the third lumbar space. This space is not below the level of the cord in many monkeys but no paralysis followed any of the inoculations.

Virulence of the organism plays an important part in the injections. Subcutaneous intra-muscular injection of large quantities of virulent growth produces only trifling effects.

The first monkey died about 18 hours after the injection of the diplococcus intra-cellularis. The surface of the brain was greatly congested and punctiform hæmorrhage existed in the superficial portion of the cortex.

The lumbar and thoracic portions of the cord were covered chiefly posteriorly with an opaque white exudate.

The diplococcus was recovered in cultures from spinal cord and brain and from the blood of the heart. Cover-glass preparation of the exudate from the brain and cord showed the organism within the polymorpho-nuclear leucocytes. Sections of the brain showed an invasion of the superficial portion of the cortex from the pia-arachnoid.

A second monkey was inoculated with the sedimented exudate from the same case with a negative result. A monkey inoculated with a smaller amount of growth survived for 43 hours; two others with larger amounts died in 14 and 15 hours.

Then follows an experiment which shows that a fatal issue may take place after a lapse of 30 hours with diminution and degeneration of the diplococcus, but without the production of any striking pathological lesion; and in contrast to this, one in which an inoculated monkey survived 12 hours and in which there was extensive meningeal infection and acute encephalitis.

These experiments would seem to show that to a certain extent

the symptoms of acute meningitis as they occur in man can be reproduced experimentally in monkeys. Not all monkeys inoculated into the spinal canal succumbed to the injections; some after apparent recovery died suddenly from causes unexplained by post-mortem examination.

Intra-muscular injection of large quantities of the diplococcus cause slight or no symptoms. To ensure a fatal outcome a diplococcus of known virulence in a sufficient dose must be injected. As compared with the doses which probably determine infection in man, those used to produce the experimental disease in monkeys are colossal. In contrast to the ease with which acute inflammation can be excited is the difficulty which attends the production in monkeys of a subacute form of meningitis.

The intra-spinal injection produces either an acutely fatal meningitis or an acute disease from which recovery takes place rapidly. By following in inoculated monkeys the changes in the cerebro-spinal canal by means of lumbar puncture the progress of the injection can be traced. Disappearance of the diplococci from the canal is a good sign; early emigration of leucocytes is also a good sign; early emigration, active phagocytosis, and dissolution of the diplococci, both within and without leucocytes, are very favourable signs.

By determining the condition of the monkey by lumbar puncture it was possible to keep a small number of monkeys in a state of subacute infection with the diplococcus for a period of several days or weeks. In the end the animals succumbed to the dose administered. In these animals the exudates were thicker and firmer and covered the base and convex surface of the brain.

In one experiment a monkey during a period of 5 weeks had 9 injections of virulent cultures, the last representing 4 agar slant surfaces. This last produced a fatal result.

The experiment demonstrates the remarkable capacity which can be developed in the monkey to dispose of culture of diplococcus injection into the spinal canal. The actively bactericidal cells and secretion are capable of destroying in a few hours prodigious numbers of the diplococcus, and agglutination of the diplococcus can take place in the canal. The final overdose of cultures causes death by poisoning, and not by infecting the animal directly. Although the pathological effects produced in monkeys are comparable with those occurring in the natural disease in man, there is no real correspondence in the relative degree of susceptibility of the two species. The quantity of an active culture required to cause marked symptoms and to bring about death from meningitis in monkeys is prodigious if compared with the number of diplococci which would suffice to produce infection in human beings.

F. E. BATTEN.

GENICKSTARREEPIDEMIE. JEHLE, *Münch. med. Wchnschr.*, 1906, (242) No. 29.

UEBER DAS ENTSTEHEN DER GENICKSTARREEPIDEMIE. (243) JEHLE, *Wien. klin. Wchnschr.*, 1906, No. 25.

DAS AUFTRETEN DER ÜBERTRAGBAREN GENICKSTARRE IM (244) REGIERUNGS BEZIRK DÜSSELDORF 1905-1906 UND IHRE BEKÄMPFUNG. KROHNE, *Klinisches Jahrbuch*, 1907, Bd. 17, H. 1.

DIE ÜBERTRAGBARE GENICKSTARRE IM STADTKREISE (245) DUISBERG UND IM KREISE RUHRORT. BAHR, *Ibid.*

IN all these papers epidemic cerebro-spinal meningitis is discussed from the standpoint of preventive medicine. Jehle's views as to the method of dissemination of the disease are that the germ has its habitat in mines, which, from their warmth and moisture, form gigantic incubators favouring its growth. Miners become infected without themselves contracting the disease, and carry the organisms to their homes, where the children in particular, being susceptible subjects, are infected and succumb to cerebro-spinal meningitis. Jehle's theory is based on the following observations: (1) In cases occurring in rapid succession in point of time it is never possible to have direct contagion from one to another. (2) Children are the principal sufferers, yet school epidemics are unknown. (3) The children who fell victims to the disease in the localities Jehle investigated belonged for the most part to families of workers in one or two mines; children of workers in other mines escaped. (4) In a district named Neumuhl there are two principal mines, and the miners inhabit separate villages. For the first few weeks of the epidemic cases were limited to one village; later the disease spread to the second. During the period in which one village only was attacked, none of its miners transferred themselves to the second mine. Early in February 1905, however, some men left the infected mine to work in the other, and in ten days the disease appeared in the corresponding village. Jehle supposes that miners are particularly likely to transmit the contagion, as they are liable to colds and catarrhal affections, and are constantly hawking and coughing. The mucus thus expelled from the nares contains the germ, and hence infection is spread. Children, on the other hand, do not cough or hawk up mucus, hence they are not so dangerous, even though they harbour meningococci or actually suffer from

the disease. Jehle asserts, therefore, that the ordinary sanitary regulations, such as closing schools, disinfection of houses, isolation of affected children, etc., are of little use as preventives; the only reasonable course, in his view, is to isolate contacts and workers in infected mines, and to disinfect their nares, for which pyocyanase is advised.

Attractive as this theory sounds, it meets with a good deal of criticism at the hands of Krohne, and as his opportunities for investigating the epidemiology of cerebro-spinal meningitis were very much more extensive than those of Jehle, we must take it that the latter has been somewhat premature in his conclusions. Krohne finds that on the whole the children of miners suffer more than those of other sections of the community. Whether, however, the mines actually harbour the organisms and act as breeding chambers for them (comparable for instance to what obtains in ankylostomiasis) is at present mere surmise, and requires much bacteriological inquiry. Even the much easier question, whether the prevalence among miners may not merely be due to the crowded conditions under which they work, irrespective of any special influence of the mine, *qua* mine, has not yet been thoroughly studied. Jehle's observations were, unfortunately, not extended either to all the cases of cerebro-spinal meningitis in the districts in question, and he did not take into account all possible factors; many cases occurred in which there was no traceable connection with mines. Jehle's material was incomplete, and his study extended only over a short period, and he is wrong in stating that the measures he adopted (isolation and disinfection of contacts) checked the epidemic. Krohne accepts the view that those who carry infection may be in perfect health though harbouring virulent meningococci; he does not for a moment admit, however, that because this is so the ordinary precautions are useless. He advises—(1) Isolation of patients, whom he regards as much more dangerous than healthy persons harbouring the organisms; (2) isolation of infected persons though healthy; (3) compulsory notification; (4) disinfection of homes and clothing; (5) preventing children from infected houses going to school; and (6) the dissemination of printed information concerning the disease. Bahr advises (1) immediate bacteriological examination of contacts, to discover the infected person; (2) on account of the rapid death of the meningococcus outside the body, local bacteriological investigation is required, as material sent to a central institution is often useless on account of delayed transit; (3) contacts who harbour organisms must be isolated; (4) patients suffering from the disease are fresh sources of infection, and must be isolated; (5) thorough disinfection of houses, etc., is essential.

J. S. FOWLER.

CLINICAL STUDIES OF THE CEREBRO-SPINAL FLUID, WITH ESPECIAL REFERENCE TO PRESSURE, PROTEIN-CONTENT, AND THE NUMBER AND CHARACTER OF THE CELLS. ROUS, *Am. Journ. Med. Sci.*, April 1907.

(246)

THIS paper records succinctly the observations of the writer upon the cerebro-spinal fluid in various conditions, under the four headings of (1) pressure in the subarachnoid space; (2) the protein-content; (3) the cell-content; and (4) the cell character of the fluid.

He estimates the pressure by attaching a vertical tube of 1·5 mm. diameter to the puncture needle, so as to measure the pressure in terms of the height of fluid supported; the fact that the vertical tube communicates freely with the subarachnoid space is shown by pulse and respiratory waves. The average normal pressure was 210 mm.; that in tuberculous meningitis rose to 550 mm., with 365 mm. as an average for all cases examined; and in purulent cases there was an average of 390 mm.

The cells were counted by means of an ordinary Zeiss red blood corpuscle pipette and counting-chamber in the following manner. A saturated aqueous solution of methyl violet is drawn up to the fourth decimal division, and the tube is then filled up with cerebro-spinal fluid. The cells are stained slightly, and are counted in the ordinary manner. The number normally present he found might amount to 7 per c.mm. In some cases of tuberculous meningitis the count reached over 600, and was never below 43, with an average of 213 per c.mm., there being a general tendency to lymphocytosis. Numerous observations and experiments proved to the writer that the so-called "clear elements," or "pseudo-endothelial cells," are in reality degenerated cells—an important point in making a cell count.

The amount of serum-albumin and serum-globulin present the writer estimates by a tube similar to, but narrower than, the Esbach tube, and the usual Esbach's solution. Normally he found mere traces, but in disease, *e.g.* tuberculous meningitis, from ·5 to 10 grammes per litre.

Detailed observations on these lines are given of series of cases in which various nervous diseases were present.

JOHN D. COMRIE.

THE MYOPATHIC PARAPLEGIA OF THE AGED. (La paraplégie myopathique des vieillards.) RONNE, *La Presse Médicale*, April 6, 1907, p. 221.

(247)

THIS is chiefly a review of Lhermitte's thesis (1907) on the above subject. Of the three groups of senile paralysis—cerebral, spinal,

and muscle—the latter is the least known, in spite of its precise picture. It usually appears after the age of 75. The early symptoms are pain and weakness. The reflexes are normal, and, at this stage, the muscles though flabby are not wasted. After the patient takes to bed, however, atrophy of the muscles soon sets in, and they feel to the hands like rigid cords. Tenderness is limited to the muscles. Contractures begin, flexion predominating. The joints remain healthy. Arterial lesions are constantly found, though Lhermitte considers that an irritative agent is probably at work, and that malnutrition of the muscles is not the sole cause of the condition.

ERNEST JONES.

THE SUBSTANCES THAT SHARE IN THE PRODUCTION OF
(248) **EPILEPTIC ATTACKS. (Die bei der Auslösung des epileptischen Krampfanfalls beteiligten Substanzen.)** JULIUS DONATH, *Deutsche Zeitschr. f. Nervenheilk.*, Bd. 32, S. 232-263.

THIS paper gives the results of a series of experiments on various animals, the object of which was to determine the capacity of certain chemical substances to produce epileptic fits.

Various urinary acids and acid salts, such as uric acid, produced no convulsions. In particular sodium biurate when injected intravenously to the amount of thirty-six times the highest percentage ever recorded in gout, had no effect whatever. Donath concludes that the urinary changes described by Krainsky and others in epilepsy—which have been controverted particularly by Hoppe—are secondary, and bear no relation to the production of epilepsy.

Ammonia salts, on the contrary, are very potent agents in the production of convulsions when injected in large doses. The same remark applies to allied bodies, such as cholin, kreatinin, trimethylamin, etc.

Lactic acid and its salts are without action. Donath also failed to demonstrate these substances in the cerebro-spinal fluid of fifteen epileptics, even when this was examined in amounts of 100 cm.

He incidentally remarks that Turner's recent findings in epilepsy (cortical hæmorrhages, excess of blood platelets, etc.) are to be regarded as secondary in nature. ERNEST JONES.

EMOTIONAL EPILEPSY, A GROUP CLINICALLY SEPARABLE
(249) **FROM TRUE EPILEPSY. (Die Affektepilepsie, eine klinisch von der echten Epilepsie abtrennbare Gruppe.)** BRATZ and LEUBUSCHER, *Deut. med. Wchnschr.*, April 11, 1907.

THE writers, as the result of a large experience of epileptics in an institution, seek to divide the cases under the heading of several diseases, not only from the point of view of causation, but as

clinically distinguishable. They separate a special group of "emotional" epilepsy from "true" epilepsy and hystero-epilepsy.

In emotional epilepsy the following general characters are found: Hereditary defects in all cases; occasional attacks of *grand mal* are observed, but more frequently there are auditory or visual hallucinations, lasting hours or days, and very frequently maniacal seizures; disturbances of mood amounting to delusions of unseen influence; in one-third of the cases suicidal attempts; and, in all, moral defects and emotional facility. On physical examination of over twenty cases, the majority showed general hypalgesia, very easily obtained dermography, and a singular pallor of the skin combined with very ready production of flushing under slight mental excitement.

The writers quote Kluge, Oppenheim, and others who have differentiated similar groups, and give a short bibliography bearing on the condition. JOHN D. COMRIE.

FURTHER CASE OF TEMPORARY ABSENCE OF THE KNEE-
(250) **JERK IN HYSTERIA. (Über einen weiteren Fall von zeitweisem Fehlen der Patellarreflexe bei Hysterie.)** WIGAND, *Neurolog. Centralbl.*, April 2, 1907, S. 293.

THE writer makes some preliminary remarks about those authors who refuse to be sceptical on the subject, but instead steadfastly believe, in spite of all the evidence to the contrary, that hysteria can never cause absence of the knee-jerk. He then adds another such case to those already described by Nonne, Dejerine, Marie, etc.

The patient, a man of 35, had complete flaccid paraplegia, there being no *voluntary* movement possible at any joint in the lower extremities. He could walk however, though with a hysterical gait. There was pronounced analgesia from the groins downwards. The electrical reactions, bladder functions, and reflexes were throughout normal with the exception of the knee-jerks. Here weakness of the left only, of both, absence of the left only, and of both, were found at different stages of the disease. The observations, which were made at Nonne's clinic, were of course carried out with all precautions, and were confirmed at different periods during the four years that the case was being watched. The illness began after severe psychical trauma six years before, and many other hysteric symptoms such as ageusia, anosmia, hallucinations, etc., were present. No sign pointed at any time to organic disease.

Hysteria can unquestionably bring about either absence or asymmetry of the knee-jerks, in spite of the current teaching to the contrary. ERNEST JONES.

CONTRIBUTION TO THE EXPLANATION OF OPHTHALMO-
(251) **PLEGIC MIGRAINE. (Beitrag zur Erklärung der ophthalmoplegischen Migräne.)** VÁCLAR PLAVEC, *Deutsche Zeitschr. f. Nervenheilk.*, Bd. 32, S. 183-231.

PLAVEC begins his comprehensive review of this subject by considering its nosological aspects. He sees in the oculo-motor paralysis of this condition something very distinct from the ordinary recurrent paralyses of this nerve with which it has been confused. Further, he supports Charcot's opinion that there is a primary disease ophthalmoplegic migraine, in opposition to Moebius and others, who maintain that it is always secondary to some or other organic intracranial affections. The pain which undoubtedly has the character of migraine as distinct from those of headache may or may not be identical with pure migraine; Plavec thinks so. The facts bearing on the above questions are mainly as follows. Ophthalmoplegic migraine always begins before twenty-five—this was so in all the thirty typical cases that have been recorded,—pure migraine does so usually. Both occur in women twice as often as in men. The former is invariably unilateral, the latter in only 50 per cent. of the cases. The former lasts usually two to three days, the latter less. The influence of heredity is more important in the case of pure migraine. The occurrence of a visual aura is commoner with pure migraine than with the ophthalmoplegic.

The migraine is to be looked upon as bringing about paralysis as its consequence; at all events, paralysis occurs in each attack only after the pain has been present for some little time. It would also seem that if migraine is going to bring about paralytic attacks it does so early in youth, so that some other factor, perhaps an anatomical one, is necessary to explain why some migrainous patients get paralysis and others never.

Both the common and the ophthalmoplegic migraine are due to a local basal affection, probably a periodic swelling of the hypophysis. In the former condition this swelling is probably due to an active hyperæmia, and in the latter to a venous stasis, which undergoes periodic exacerbation. The writer suggests the existence of a partial lateral dislocation of the hypophysis in the latter cases, thus accounting for the pressure on the oculo-motor nerve in the cavernous sinus. ERNEST JONES.

SOME UNUSUAL OCULAR MANIFESTATIONS OF ARTERIO-
(252) **SCLEROSIS.** W. ZENTMAYER (of Philadelphia), *Journ. of Am. Med. Ass.*, March 2, 1907.

ONE of the first to describe the effects on the optic nerve of sclerosed vessels at the base of the brain was Michel, in 1877.

His case showed bilateral choked disc, and he assumed that it was caused by pressure on the lymph spaces of the optic nerve. In 1893 Otto presented a series of such cases and drew some conclusions as to the nature of the anatomical changes and their clinical manifestations. Bernheimer (1891), Liebrecht (1902), and Bull (1904) added evidence to support the claims of previous writers.

Zentmayer believes that in the earlier stages of arterio-sclerosis of the smaller vessels, spasm or collapse of the ophthalmic branches may occur and is often the explanation of sudden blindness occurring in these cases. Other writers claim that because of the free anastomosis of vessels at this point such a theory is untenable. Von Graefe (1869), Rothmund (1866), and Loring (1874) published cases in which they explained this clinical manifestation as being due to "spasm of the central artery of the retina." In Von Graefe's case, operation (irridectomy on one eye and paracentesis on the other) relieved the condition. The author gives in careful detail the history of a case in which he feels certain that spasm of the central artery of the retina repeatedly occurred while under his observation.

Concerning the prognosis in such cases of transient blindness, a distinction should be made between those where the attack is but a symptom of a general condition such as epilepsy, Raynaud's disease, migraine, etc., and those where it is a local manifestation of a general vascular disturbance. In the former class the prognosis for recovery is much better than in the latter. In regard to treatment, drugs which will reduce arterial tension are recommended; a few selected cases may be benefited by operation.

C. H. HOLMES.

(253) **CONJUGATE DEVIATION OF THE EYES AND HEAD AND DISORDERS OF THE ASSOCIATED OCULAR MOVEMENTS.** T. WEISENBURG (Philadelphia), *Journ. of Am. Med. Ass.*, March 23, 1907.

THE author reviews with painstaking care some of the literature on this subject, and adds his own results from a study of sixteen cases in which there was conjugate deviation of the eyes and head. Fifteen of the cases were with necropsy. He draws the following conclusions:—

1. Conjugate deviation of the eyes and of the head is dependent on a most complex mechanism.

2. In the human being there is but one oculo-motor centre, or at least one active functionating centre, situated in the posterior portion of the second and third frontal convolutions, adjacent to the precentral convolutions.

3. A separate centre exists for the movements of the head, probably in the lower anterior portion of the precentral convolutions.

4. There is probably in man a distinct centre for the combined movement of the eyes and head, situated in the area between the head and eye centres.

5. It is probable that the cortical oculo-motor, head, and combined head and eye centres are subdivided for lateral as well as for upward and downward movements.

6. The oculo-motor and the motor head centres are in connection by means of association fibres with the cortical centres for the special senses, in the temporal, occipital, uncinate, and other lobes.

7. Any lesion in the motor centres for the eyes and head, or in the related special sense centres, or in the association fibres connecting the former with the latter, will cause an impairment in voluntary deviation of the eyes or of the head, or of both, this depending on the nature and location of the lesion.

8. Lesions in the angular gyrus cause conjugate deviation because of involvement of visual and auditory fibres which lie underneath this area.

9. The theory of Bard that conjugate deviation of the eyes and head is always, or nearly always, accompanied by homonymous lateral hemianopsia and dependent on this is an error, for in the majority of instances hemianopsia does not exist.

10. Hemianopsia may be caused by the shock or transient effects of the hæmorrhage, this loss of half-vision being only a temporary symptom, and similar in nature to the temporary hemianæsthesias sometimes observed in capsular lesions.

11. The occurrence of conjugate deviation of the eyes or of the head, or of both, is of no value as a localising symptom, because it may be the result of a lesion in any portion of the cerebrum. It may be of value, however, in conjunction with other localising symptoms.

12. Conjugate deviation of the eyes and head probably occurs in every case of large apoplectic lesions, but in some instances it is a partial or minor form, and of transient duration.

13. Paralysis or impairment of associated ocular movement may occur as a result of a hemianopsia. This, however, is only temporary.

14. There is at present no evidence of a centre or centres for automatic ocular movements, but if such centres exist in the thalamus, a lesion of these should cause forced or inco-ordinate ocular movements.

15. Convergence and divergence are probably not reflex acts, but associated ocular movements similar to lateral and upward movements. They probably have cortical centres in the posterior

portion of the second and third frontal convolutions. The movements of convergence and divergence are probably brought about by associating tracts in the pons and cerebral peduncles.

C. H. HOLMES.

CONSEQUENCES OF CRANIAL INJURY. HARVEY CUSHING, *New* (254) *York Med. Journ.*, Jan. 19 and 26, and Feb. 2, 1907.

As the basis of the paper under consideration, Cushing has selected three clinical histories, illustrating respectively the effects of extra-dural, sub-cortical, and intermeningeal hæmorrhage, and in his discussion of the points raised by each case he deals with a number of interesting and important problems.

The first case was one of fracture of the vault and base of the skull, resulting in compression, without previous concussion, due to middle meningeal hæmorrhage. When seen the patient presented the symptoms of advanced compression, with focal symptoms. These were a relative dilatation of the left pupil, slight left-sided ptosis, slight paresis and occasional twitchings of the right side of the face, a tendency to conjugate deviation of the eyes to the left, and paresis of the right arm. In discussing the value of focal symptoms in such cases, Cushing lays stress on the value of examining the optic discs, and states that this has proved of the utmost aid in his recent traumatic cases, in disclosing œdema from increased tension, and in determining the side on which primary compression is being exercised. At the operation a large osteoplastic flap was turned down on the left side, and a large clot found and removed from the temporal region. Its place had to be partly filled for two days by a gauze pack to control hæmorrhage in the region of the foramen spinosum. On removal of the gauze the patient rapidly improved, and ultimately left hospital in four weeks, almost well. In discussing the operative technique employed, Cushing states that the most useful operation in such cases is the removal of bone in the temporal region through a muscle-splitting incision, and not an osteoplastic flap operation. The intermusculo-temporal operation gives access to the mid-meningeal artery, and can be extended at will, it allows of drainage being employed, and the subsequent bone defect is well covered by the temporal muscle. Cushing believes firmly in the value of a "decompressive" operation on these lines in cases of head injury showing any signs of increased tension, as he maintains that patients after it are much less liable to suffer from post-traumatic neuroses, so common after head injuries. The patient in the case detailed remained free from any such disturbance, but developed an arterio-venous aneurism, with consequent exophthalmus on the right side. The right internal carotid artery was tied for this without relief.

The second case is one of great interest as throwing light on the question of the localisation of sensory function. The patient received a stab wound on the left side of the head just behind the Rolandic area, resulting in a localised subcortical hæmorrhage in the region of the supra-marginal and angular gyri. The symptoms were briefly, almost complete right-sided sensory paralysis of the sense of touch, pain, and temperature, with complete right-sided astereognosis and visual word-blindness. At the operation the clot was found, accurately mapped out, and removed. The cortex round the fissure of Rolando was electrically stimulated, and the results confirmed those obtained in many other cases by Frazier, Krause, Cushing, and others in showing that the excito-motor cortical centres lie entirely in front of the Rolandic fissure, as shown in anthropoid apes by Sherrington and Grünbaum. After operation the patient rapidly and completely recovered, the astereognosis being the last sign of injury to disappear. A number of interesting points in operative technique and in cerebral localisation are discussed in connection with this case.

The third case was one of epilepsy following the lodgment of a bullet in the brain; operation was done for its possible relief and the bullet was found and removed, and adhesions separated which had apparently resulted from the organisation of an intermeningeal clot. Only temporary benefit followed. In regard to the prospects in operating for focal epilepsy, Cushing states that in a majority of cases the seizures are lessened in intensity; that in a small minority they do not recur at all. Efforts to close bone defects by osteoplastic operations and to prevent adhesions re-forming by introducing foreign substances he regards as misdirected and valueless. In connection with this case Cushing again insists on the value of an exploratory operation in cases of possible subdural hæmorrhage from basal or other fracture, in order to lessen the dangers of firm adhesions forming and resulting in epilepsy, etc. These adhesions result, he believes, from the organisation of thick clots, and while a temporal operation on one or both sides will not reach all extravasations, it is most likely to yield good results.

Many other interesting questions are raised and discussed in this and the preceding cases, and the paper as a whole shows well the advances made in cranial surgery in recent times, to which Cushing has in no small degree contributed.

J. W. STRUTHERS.

(255) **CASE OF INJURY OF FRONTAL BRAIN REGION, FOLLOWED BY LOSS OF THE HIGHER MENTAL CONTROL.** ROBERT JONES, *Archives of Neurology*, Vol. iii., 1907, p. 369.

THE consensus of expert medical opinion to-day supports the view that the frontal lobes are the seat of the highest functions

of the mind. The following case, briefly stated, tends to strengthen this opinion.

X. Y., aged 26, was admitted to Claybury from prison. The family history was singularly free from insanity and drink. The patient was in good health and condition, and the organs apparently normal. The diagnosis was "organic dementia, secondary to injury of the brain," traumatism being the assigned cause of insanity, patient having fallen 40 feet down a lift and fractured his skull in the pre-frontal region. The forehead showed a linear scar, six centimetres long, with obvious depression of the right frontal bone. Patient was unconscious for several days after the accident, and there was some loss of brain substance. Six months later some portions of dead bone were removed at St Bartholomew's Hospital, and after two or three years of medical treatment he was pensioned as unfit for further work in Woolwich Arsenal. Before the accident patient was bright, energetic, honest, and trustworthy, a life abstainer, and much respected. After the accident there was a complete metamorphosis; his career became a record of moral obliquity and mental perversion. He was idle, irritable, threatening, and violent; he was three times convicted of indecent behaviour, the last time being detained during His Majesty's pleasure, and after being in prison a month was removed to Claybury. Colonel A., Royal Artillery, states: "His character has altogether changed since the injury. From being a steady, honest, and reliable man he became in every way the reverse."

A similar case is referred to in a paper by Dr Bernard Hollander in the *Journal of Medical Science* for April 1901, and known as "the American Crowbar Case," in which the highest attributes of mind and the moral nature became perverted, but without any impairment of motor or sensory functions.

AUTHOR'S ABSTRACT.

(256) **CLINICAL CONTRIBUTIONS TO THE PHYSIOLOGY OF THE SYMPATHETIC NERVOUS SYSTEM. (Klinische Beiträge zur Physiologie des sympathischen Nervensystems.)** L. R. MÜLLER, *Deut. Archiv. f. klin. Med.*, Bd. 89, H. 5-6, S. 432.

THIS is a somewhat diffuse paper, dealing mainly with our ignorance of the physiology of the sympathetic nervous system, but insisting on its dependence on the central cerebro-spinal nervous system. This is illustrated by the wide-spread influence which mental states and emotions have on almost every function of the body. Numerous examples of this are cited, but there is nothing strikingly new, nor any explanation of how the mental processes affect the working of the sympathetic system.

J. H. HARVEY PIRIE.

THE DIFFERENTIAL DIAGNOSIS OF OTITIC SINUS THROM-
(257) **BOSIS.** F. Voss (Riga), *Archives of Otology*, Dec. 1906.

In this paper, which is a translation of an article from the *Zeitschr. f. Ohrenheilk.*, vol. l., the author points out that an accurate diagnosis is important, because, if a mistake be made, a useless operation is performed on a patient already extremely ill.

The following conditions may give rise to difficulty in diagnosis:—

1. *Malaria.*—In both the pyæmic and septicæmic forms of sinus thrombosis the spleen may be enlarged, and this may cause an error in diagnosis, especially in malarial districts; in malarial cases, however, the spleen is hard. A previous history of ague is important, and a case is quoted which showed chronic purulent otitis media, headache, chills, rapid rise and fall of temperature, marked perspiration, and a hard, palpable spleen: a previous history of malaria was obtained, and the condition cleared up on administration of quinine. First attacks of malaria in patients with purulent otitis media are most difficult to diagnose, as in such cases the ague is atypical. One such case, with mastoid tenderness, got well on the administration of quinine, though the plasmodion was not found in the blood and the spleen was not enlarged. Voss, however, regarded the case as one of malaria on account of its reaction to quinine.

Another case is reported of chronic purulent otitis media during pregnancy, in which there were daily rigors for four days; no mastoid swelling or tenderness and no optic neuritis; the spleen was large and hard but not tender; plasmodia not found. Quinine failed to prevent the rigors. At the operation, cholesteatoma was discovered, the sinus wall was necrotic, and the contents thrombosed; the case aborted but eventually recovered. The splenic enlargement did not disappear till two months after the operation. This is the only sinus case in the author's experience in which the spleen was large and hard but not tender.

2. *Tuberculosis.*—Two cases of pulmonary tuberculosis resembling sinus thrombosis are referred to: in the first the patient suffered from pain in the ear five days after taking measles, and a week later a rigor occurred; paracentesis evacuated no exudate; pulmonary catarrh was present and a few tubercle bacilli were found in the sputum; the otitis rapidly cleared up, but the patient died a year later (presumably from pulmonary tuberculosis). The other case was one of otitis media purulenta with total loss of both drums due to scarlatina: on the twenty-fifth day rigors began with remitting temperature; death three months later; the autopsy showed disseminated tuberculosis; meninges and sinus free.

3. *Septic Endocarditis* has the same symptom-complex as sinus thrombosis, and this makes the examination of the eyes for hæmorrhages all the more important.

4. *Hysteria.*—Voss has seen a case of hysteria with normal drum and hearing mistaken for sinus thrombosis, and operated on by another surgeon. Even after this operation the patient still claimed to have rigors, though her temperature was normal; she was anxious for further operative interference.

5. *Puerperal State.*—Two cases of purulent otitis media are mentioned as a complication of this condition: the first turned out to be one of sinus thrombosis and resulted fatally; the second was a local (probably perineal) condition and ended favourably.

6. *Typhoid Fever.*—The difficulty in diagnosing otitis media occurring in the course of typhoid fever from sinus thrombosis with continuous high temperature is next touched on; splenic enlargement occurs in both conditions. Bezold, however, states that otitis due to typhoid fever rarely begins before the third week of the disease. If, therefore, the acute otitis begins first and the high temperature occurs later with increased pain in the ear and head, the diagnosis of sinus thrombosis is likely.

7. *Pneumonia.* — If this disease has already been diagnosed before otitis media begins the difficulty is not so great, but even then, if the temperature varies or if rigors occur, the "physician is apt to believe that it must emanate from the ear." A case is mentioned in which there were rigors and otitis media, but no perforation of the drum: the case eventually got well without perforation or paracentesis.

8. *Erysipelas.*—Four cases of acute otitis media with rigors and high fever have been sent to Voss for operation: erysipelas of the scalp was present, involving the mastoid process and simulating mastoiditis.

9. *Antipyretics.*—Voss has seen several cases of typhoid fever complicated with otitis media, in which the administration of pyramidon gave rise to difficulty in diagnosis: this drug caused a fall of temperature, but every time the temperature rose again a rigor occurred; when the drug was discontinued the temperature curve characteristic of typhoid returned. J. S. FRASER.

MEDICAL THROMBOSIS OF THE SINUSES OF THE DURA MATER. (258) J. BERTIER, *Arch. Gén. de Méd.*, April 1907.

THE writer distinguishes between surgical thrombosis, which is usually due to some local cause, suppurative in nature and limited in extent, and medical thrombosis, which arises from general blood states or causes acting from a distance, is not suppurative, and is widespread in character. In infants the most frequent causes are

gastro-enteritis and broncho-pneumonia, especially when complicating one of the infective fevers, but marasmus, kidney disease, and cachexia, associated with some form of tuberculosis, are also of etiological importance. In adults chlorosis acts as the most important predisposing factor, and among other causes are the acute exanthemata, puerperal and other infections, and tubercular cachexia. It is probable that a lesion of the endothelial lining of a vein is essential to the formation of a thrombosis. In most cases the cause is undoubtedly of an organismal nature, but one cannot affirm that all are due to an infective agent. The sinuses of the brain are predisposed to thrombosis by their position in the circulatory system, and by their anatomical formation. The symptoms produced are the result of cortical irritation and softening, brought about by venous stasis and œdema. In infancy there are usually convulsions and rapid rise of temperature, and in later childhood delirium and paralyses are superadded. In the adult the symptoms are much more complex. Violent headache, followed at varying intervals by cerebral vomiting, vertigo, epileptic seizures, paralyses, delirium, and coma would constitute a typical case resulting from severe chlorosis. Hyperpyrexia just before death is an important symptom. In the majority of cases the disease is fatal within two or three days, but fibrous organisation of the clot, with consequent hydrocephalus or paralyses, may occur in some cases, and it seems certain that absorption of the thrombus with complete recovery may occasionally take place. The diagnosis is always difficult, and must, in most cases, be made by a process of exclusion, careful search being made, especially in the adult, for any possible etiological condition. The differential diagnosis has to be made from simple convulsions in infancy, tuberculous meningitis, cerebro-spinal meningitis, hysteria, uræmia, eclampsia, tumour, and other conditions. The literature of the subject is given in full at the end of the article. HENRY J. DUNBAR.

PSYCHIATRY.

(259) **TWO CASES OF AMAUROTIC DEMENTIA (IDIOCY) AND A CORRELATION OF THE MICROSCOPIC CHANGES IN THE CENTRAL NERVOUS SYSTEM WITH THE RESULT OF A CHEMICAL ANALYSIS OF THE BRAINS.** F. W. MOTT, *Archives of Neurology*, Vol. iii., p. 218.

THE special interest of this paper lies in the fact that, in addition to a very complete and careful histological examination, portions of the brains of Dr Mott's two cases were submitted to a chemical

analysis, with the result that the interpretation of the anatomical changes was confirmed.

The clinical histories of the two cases offer no points of special interest; both were typical of the disease. The first case was a female child, the offspring of Russian Jews, who at the age of 12 months was found blind and suffering with progressive weakness of the limbs and rigidity, which had an insidious onset; later there was complete paralysis except of the automatic functions of sucking, swallowing, and breathing, and hardly any sensory response to the surroundings. The child lay apathetic and helpless, but it was only in the last stages of the illness that any wasting of the muscles became visible. There were frequent convulsions. The knee-jerks remained present and the plantar reflexes were of the extensor type. Ophthalmoscopical examination revealed the presence of a typical dark brownish-red spot at the macula, surrounded by a pale zone. The child died at the age of 1 year and 10 months. In the second case the disease was less far advanced, the child died at the age of 11 months.

The convolutional pattern was fairly good in each brain, but in both the cortex was tough and leather-like in consistence, and in the case in which the disease was further advanced the brain was abnormally heavy, due to a hypertrophic gliosis. Microscopical examination revealed the changes in the nerve cells which must now be regarded as typical, if not pathognomonic, of the disease. Its essential histological feature is a progressive loss of the Nissl substance of all the neurons, and it was ascertained in the second case, for the first time, that it is not merely the cells of the central nervous system, but that also those of the sympathetic ganglia are involved. The mode in which the Nissl substance disappears is progressively from the periphery of the neuron towards the nucleus in the centre of the cell. It may be, therefore, assumed that the Nissl substance is a derivative of nuclear biochemical interaction on the cell protoplasm and its lymph environment. The cause of this metabolic metamorphosis may be an inherent lack of specific energy, racial or familial, of the neurons, and possibly some hitherto undiscovered bio-chemical alteration of the blood or lymph. The wide-spread nature of the disease, not a single nerve-cell in the body seems to escape, argues in favour of the theory that the disease is due to some such metabolic disturbance and not to inborn morbid anatomical changes. The changes in the nerve-fibres and of the neuroglia are secondary to that of the cells. The loss of myelinated fibres seems to be proportional to the duration and the severity of the disease; in the first case the central white matter of the hemispheres was semifluid and like cream in places, and on examination by Weigert's method considerable deficiency of these fibres was detected, especially in

the horizontal layers of the cortex. Weigert's neuroglia stain revealed glial sclerosis throughout the whole nervous system, but especially in the cortex, and there was in places some perivascular cell infiltration due to proliferating endothelial cells. Practically all the ganglion cells had disappeared from the retinæ.

An important observation was the evidence of a chronic inflammatory process in the pancreas, adrenals, liver, and the wall of the intestinal tract, and in these organs many of the functional cells stained badly, and were represented only by swollen nuclei surrounded by little or no protoplasm.

As the disappearance of the Nissl substance, which is chemically a nucleo-proteid, is the predominant change that is revealed by histological examination, the disease must be regarded as primarily a failure in the elaboration of the nucleo-proteid of the neurons. This was confirmed by the chemical analysis of portions of the two brains, by which it was shown that there was a great decrease in nucleo-proteid corresponding to the loss of the Nissl substance, and an increase in simple proteid which may be correlated with the increase of glial fibres.

GORDON HOLMES.

THE MECHANISM OF THE ELECTRICAL REACTION OF
(260) **JOLLY**. ALBERTO SALMON (Firenze), *Polioclinico, Sez. Med.*, Vol. xiv., 1907.

ON account of its peculiarities, which have been well defined by Jolly, Murri, Goldflam, Flora, and Grocco, the reaction of Jolly should be distinguished from the myasthenic reaction properly so-called, of muscular origin, which is found in the primary myopathies and in certain muscular atrophies. In the myasthenic reaction we find electrical muscular exhaustion, not only to the faradic current, but also to galvanic stimuli. Borghesini of Padua has recently described a case of myasthenia in which histological examination of the muscles revealed a very profound lesion of the striated fibres, and in this case a prompt galvanic myasthenic reaction was noted. The reaction of Jolly is, on the contrary, characterised solely by the rapid loss of the tetano-faradic contraction; the muscles, although apparently exhausted, react perfectly to voluntary and galvanic stimuli; even the muscular inexcitability to faradism is limited to the points directly excited by the electrode, no influence being exercised on the excitability of the adjacent points of the muscle or corresponding nerve. And finally, the variability of the reaction is characteristic, its relations to the asthenia not being always constant (Grocco). If the definition "myasthenic reaction" is very exact as applied to the electrical

modifications observed in the first case, one cannot say the same with regard to the reaction of Jolly, in which we find normal excitability of the peripheral motor apparatus. Indeed the muscle, apparently exhausted by the faradism, gives a prompt and vigorous contraction with the galvanic stimulation. Galvanism not only is powerless to produce the reaction of Jolly, but it has a favourable influence upon faradic exhaustion (Murri). Moreover, in the reaction of Jolly there cannot be exhaustion of the intramuscular motor nerve terminations (*placche motrici*), since we find that the muscle exhausted by faradism responds perfectly to indirect stimulation by the nerve.

The reaction of Jolly, as observed on faradisation of the peripheral nerves (Goldflam, Murri), cannot be explained by their exhaustion. To this conception is opposed the perfect excitability of the intramuscular nerve terminations, which we know always become exhausted before the nerve, as well as the fact that in the reaction of Jolly the nerves, even at the points which cannot be exhausted by faradism, respond normally to galvanic stimuli. We must therefore suppose a qualitative change of the exhaustive nerve to the two electric stimuli, which is very rarely observed in affections of the nerve. The mechanism of this interesting reaction, however, would be perfectly cleared up by supposing that the cutaneous nerves, which are the best conductors of faradism in the skin, become fatigued and rapidly lose the property of transmitting faradic stimuli to the neuro-muscular motor apparatus, whence its unexcitability.

A very valuable symptom in myasthenia is the fatigue of the cutaneous nerves to faradism, as evidenced by the *rapid exhaustion of the farado-cutaneous sensibility.* This symptom was observed by Flora, Albertoni, and by the writer in three cases with reaction of Jolly. Our hypothesis would explain the peculiarities of the reaction of Jolly otherwise inexplicable. These are:—

1. The loss of excitability of the nerves and muscles is limited to the cutaneous points directly excited by the electrode.

2. The exhaustion obtained by faradism on the muscle in on way modifies its excitability through the nerve, and vice versa.

3. The independence of the reaction of Jolly and the asthenia, frequently observed, and its presence in the muscles which retain perfectly their muscular tonus.

4. The absence of influence of the farado-muscular exhaustion upon the voluntary and galvanic stimuli.

The reaction of Jolly, essentially due to fatigue of the cutaneous nerves to the faradic current (tetano), very probably owes its origin to the functional depression of the cortical centres which preside over the nervous apparatus of the skin. The cortical origin of the reaction of Jolly, already sustained by Goldflam, would accord

perfectly with the clinical observation which shows the frequency of the electric phenomenon in neurasthenia, hysteria, the traumatic neuroses, and in Erb's disease—affections which are very frequently associated with serious exhaustion of the cortical sensorio-motor centres. AUTHOR'S ABSTRACT.

HERPES OF THE AURICLE, WITH NEURITIS OF THE FACIAL
(261) **NERVE.** T. SARAI (Japan), *Archives of Otology*, Dec. 1906.

THIS is a translation of an article in the *Zeitschr. f. Ohrenheilk.*, vol. xlvi., which refers to previous cases noted by Gruber, Politzer, and others. In the case reported the patient was suddenly seized with pain in and in front of the left ear, and on the following day left-sided facial paralysis appeared. The sense of taste was lost in the anterior third of the left half of the tongue, but the palate was normal; the concha showed a vesicular eruption; the canal, drum, and hearing were normal; the pain disappeared in two days and the herpes eleven days later, but the paralysis persisted for five months, despite electrical treatment. Gruber has pointed out that the condition differs from herpes of the intercostal nerves, because the eruption is situated in the distribution of the auriculo-temporal, not in that of the facial nerve. He suggests that the condition may be one of neuritis of the former nerve, which sometimes communicates with the facial. J. S. FRASER.

A CASE OF INTERMITTENT ANGIOSCLEROTIC DYSKINESIA
(262) **OF THE ARM.** **(Ein Fall von Dyskinesia intermittens angiosclerotica brachii.)** STENDER, *St Petersburger med. Wchnschr.*, No. 4, 1907.

WHILE intermittent limping has been long recognised as a symptom connected with arterio-sclerosis, Determann and Erb have shown recently that a similar condition obtains in other parts of the body like the tongue and arm, and the present writer records a case affecting the arm.

The patient, a washerwoman, aged 50, had suffered for three years from "tearing" pains in the region of the right shoulder whenever she made great or protracted efforts with the right arm. These always passed off after she had desisted from work and rested the arm for twenty or thirty minutes, and came on again after a similar period of work. In this way her daily work consisted of an alternation of periods of activity and of rest. On examination the heart was found to be hypertrophied and the aortic valve insufficient, the radial arteries were hard; in the right radial the pulse was small and regular, while the left showed

the typical characters of aortic incompetence. Two months after she came under observation the right pulse ceased to be perceptible even at rest.

The writer attributes all the symptoms in these cases to a loss of power to regulate the proper distribution of blood to various parts according to their functional needs, in consequence either of arterial spasm or sclerosis.

JOHN D. COMRIE.

A CASE OF CHRONIC BAD SPELLING—AMNESIA VISUALIS
(263) **VERBALIS—DUE TO ARRREST OF POST-NATAL DEVELOPMENT.** LIGHTNER WITMER, *The Psychological Clinic*, Philadelphia, Vol. i., No. 2, p. 53.

AN intelligent boy at fourteen had only reached the standard attained by other boys at eleven. He had thus lost three years—more than one-third of his school life. The cause of his retardation was an inability to acquire the spelling of words, and a consequent inability to read. The only words he could pronounce without spelling were a few monosyllables of not more than three letters. He had a good memory for sounds, and a good visual memory for colour and simple geometrical figures and even for separate letters. Vision of each eye was practically normal.

The author attributes this condition to a want of ability to co-ordinate the movements of the eyes. This inability was not sufficient to cause any apparent squint, but was sufficient to cause diplopia. The visual perception was improved by wearing proper glasses.

Visual perception having thus been improved, the attempt was made to educate the cerebral centres by instruction in rapid reading, in order to induce him to "jump" at words without previously spelling them. At the end of six months there was considerable improvement, and he was beginning to read at times for pleasure. At the age of seventeen the boy reported that he had not failed of promotion at school since his special training began, that he did not find reading specially difficult, and that he was fond of Cooper's novels. Four years later the patient developed tuberculosis, of which he died.

This case is obviously closely related to the cases which Hinshelwood and others have described as congenital alexia or congenital word-blindness, and which are usually ascribed to a failure of the centres concerned in reading to develop. In this case there was certainly such a failure, but the interesting question is whether this was due to some congenital insusceptibility to training, or whether the ocular defect alone is a sufficient explana-

tion. The author states that in his experience chronic bad spelling is invariably associated with some form of defective vision, but he says he is not convinced that the bad spelling is necessarily caused by the eye defect.

It is difficult to come to a conclusion as to this point. We would like to suggest that some light might be thrown on the question by the child's ability to learn arithmetic. Acquired alexia may be associated with the retention of ability to read figures, and in some of the published cases of congenital alexia progress in arithmetic was normal. In such cases as Dr Witmer's one would expect to find, if a visual defect were the sole cause of the arrested development, that arithmetic would be defective as well as reading. Probably the recognition of figures may be more easily attained than the recognition of words, but a diplopia which prevented *any* progress in reading would scarcely be compatible with normal progress in arithmetic. Dr Witmer tells us that his case made good progress in his school subjects other than reading, but he does not specially mention arithmetic.

W. B. DRUMMOND.

TREATMENT.

RADIOTHERAPY OF SYRINGOMYELIA. (La radiothérapie de la (264) **syringomyélie.)** BEAUJARD and LHERMITTE, *La Semaine Médicale*, April 24, Année xxvii., p. 193.

THIS article is a complete review of the subject, and contains in addition an account of four additional cases all treated by radiotherapy. One of these was treated thus once a week and carefully observed for eleven months. The results of this method of treatment, which was first introduced by Raymond, were very encouraging in every case. The improvement was seen chiefly in the motor symptoms, though the sensory and trophic disturbances also diminished; sensation returned in the inverse order to its loss, so that touch improved more than sensibility for pain and much more than that for temperature. When atrophy had ensued of course the improvement was slight.

The authors speculate as to the action of the X-rays in syringomyelia. They remind us of the resemblance of the lesion to a neoplasm, which is frequently affected by the action of X-rays. The symptoms improve to an unequal degree, because they are of dissimilar pathogeny. Destruction, pressure, œdema, etc., all play their share in the production of symptoms, and when retrogression of the lesion occurs these processes will be unequally influenced.

The details of technique are fully discussed.

ERNEST JONES.

TREATMENT OF EPILEPSY. (Contribution à l'étude du traitement de l'épilepsie.) HEYNINX, *Journ. de Neurologie*, March 20, 1907, p. 101.
(265)

THIS paper, based on four observations, has as its main thesis the importance of nasal infection in the production of epilepsy. The four cases, in a girl of 11 and men of 18, 23, and 25 respectively, are communicated in great detail. In every instance great improvement, if not cure, followed operative treatment of the nasal condition.

The author suggests that toxins, without actual microbes, penetrate from the nasal to the cranial cavity, and there exert their noxious effect on the cerebral cortex. Without explicitly saying so, he gives the impression that he considers the majority of epilepsies to be due thereto.

At the discussion of the paper, which was read before the Belgian Society of Neurology, the opinions expressed by the members, including Decroly and Sano, were on the whole very favourable. ERNEST JONES.

Reviews

MANIC-DEPRESSIVE INSANITY. (La psychose maniaque-dépressive.) Dr G. DENY and Dr PAUL CAMUS. Pp. 96, 10 Illustrations. Paris: J. B. Baillière et fils. 1907. Price fr. 1.50.

THE modern trend of psychiatry in the direction of considering and classifying the insanities according to their whole course and termination rather than their symptomatology at particular periods in their development, and the consequent inclusion of cases having apparently different clinical characters within any given category, owes very much to the work of Kraepelin. The authors of the above interesting monograph on manic-depressive insanity maintain that the honour of initiating this mode of regard belongs to France, and the brief historical review which they give supports this contention with regard to this psychosis. That the periodic insanities, intermittent, recurrent, alternating, circular, *folies à double forme*, etc., are only different modalities of the same fundamental malady is of course not new; the novelty of the modern trend consists in the tendency to class with manic-depressive insanity the cases formerly considered cases of idiopathic mania, and to apply the term melancholia only to cases of depression in the period of involution. Manic-depressive

insanity thus becomes a very widely inclusive category, those affections really to be distinguished from it being, according to Drs Deny and Camus, "certain syndromes of the grand neuroses, such as neurasthenia, hysteria, and epilepsy; certain psychoses of toxic or infective origin, alcoholic delirium and mental confusion in particular; and finally some organic psychopathies, amongst which may be included the melancholia of involution, general paralysis, and dementia præcox." They define this form of insanity as "a constitutional psychosis, essentially hereditary, characterised by the repetition, alternation, juxtaposition or co-existence of states of excitation and depression"; and further show that it affects preponderatingly the female sex, and that it exists in the proportion of from 10 to 15 per cent. of the annual admissions to asylums. It is a disease, they say, whose phenomena are always *hypo* or *para* and never *hyper*, that is that the *hyper* phenomena are only apparent, resulting from the removal or weakening of higher inhibiting function. Finally, it is a disorder which never ends in dementia, recovery occurring in the immense majority of cases, but with an almost fatal certainty of repeated relapse. This, if a fact, is of importance as in itself justifying the adoption of manic-depressive insanity as a separate clinical category.

R. CUNYNGHAM BROWN.

THE PROGRESSIVE EVOLUTION OF THE STRUCTURE AND FUNCTIONS OF THE VISUAL CORTEX IN MAMMALIA.

F. W. MOTT, *Archives of Neurology*, Vol. iii., 1907.

IT is difficult in the space allotted to a review to do justice to this remarkable paper, which in forty-eight pages, many of which are occupied by illustrations and descriptions, contains a wealth of information such as is rarely comprised within the limits of a lengthy monograph. The writer therefore purposes to abstract, rather than to critically examine, the contents.

The paper is sub-divided into six sections, some of which, probably for reasons of space, are so condensed that reading is difficult and abstraction is almost impossible, if the important views enunciated by the author are to be clearly expressed.

Section I. deals with "the fundamental principles underlying progressive evolution of structure and function."

In this section the author, employing the terms introduced by Elliot Smith, distinguishes between the earlier portion of the pallium, or *archi-pallium*, which is functionally connected with the sense of smell and is represented in all the mammalia—"in some of the lower species, *e.g.* the insectivora, in which it forms the great bulk of the hemispheres"—and the phylogenetically newer

cerebral cortex or *neo-pallium*, which subserves "the functions of vision, hearing, and tactile kinæsthetic senses and their associated memory."

He then points out that the distinctive feature of the human cerebral hemispheres is a general cortical expansion, especially of the neo-pallium, and that this increase of territory is not so much around the primary fissures,—sylvian, central, and calcarine,—as in the frontal, temporal and parietal lobes.

The section concludes with a statement of the purpose of the paper, namely, "an endeavour to show what part vision, as a directive faculty, has played in this great increase of the neo-pallium in mammals from the lowest types up to man."

In Section II. "the structural and functional evolution of the visual cortex in mammals" is described. Dr Mott accepts, as a general basis of cortical structure, the following five layers: (1) zonal layer, (2) pyramidal, (3) granules and stellate cells, (4) inner line of Baillarger, with large solitary cells of Meynert, (5) polymorph layer. He then proceeds to describe the visual cortex in seven orders of mammals.

In the *Insectivores* the cell elements of the visual cortex consist simply of a thin layer of cells like granules (3), and of a deeper layer of polymorph cells (5). In these animals vision is rudimentary, and hardly serves as a directive faculty.

In the *Rodents* the visual cortex is more complex, and a definite thin layer of cells (2) lies above the granule layer (3). These animals possess panoramic vision of very extensive range, and the eyes are set laterally.

The author has examined two *Marsupials*, the herbivorous Wallaby and the carnivorous Dasyura. In the former the pyramidal layer (2) is imperfectly represented, and in the latter it is well defined.

In the *Ungulates* the cortex, owing to the relatively large size of the animals examined, is of considerable depth. There is a fairly-developed pyramidal layer (2), though this layer is not developed to the same extent proportionally as the other layers. These animals possess vision which is mostly panoramic and extensive in range, and also a variable degree of binocular vision.

In the *Carnivores*, with eyes set further apart than the Felidæ, the pyramidal layer (2) is of fair depth; and in the Felidæ, which possess binocular stereoscopic vision, the striking feature of the visual cortex is the depth of this layer (2). Dr Mott remarks: "The correlation of the directive faculty of vision and the executive faculty of the paw is only possible to an animal capable of converging its visual axes, and necessitates, consequently, a suitable setting of the eyes in front of the face, not too far apart, and with the nose not intervening." He adds: "We thus see, even at

this stage of the animal series, a simultaneity in the progressive development of the anatomical substrata of the visual, directive, and tactile motor executive faculties, as exemplified in the former by the marked development of the pyramidal higher associational layer of the visual cortex."

In the *Lemuridæ* the pyramidal layer (2) is developed to about the same extent as in the cat. In these animals, however, the eyes are set wide apart, and convergent vision is not possible.

It may here be remarked that beautiful illustrations of these several cortices are inserted, and these amply bear out the argument of the author. Dr Mott, however, does not state from what region of the cerebra the sections were taken, but simply refers to them as "visual cortex."

The author then refers to the visual cortex of the *Primates*, and he here deals both with the two laminations of the visual region and with the superficial extent of the cortex occupied by the visuo-sensory area. In describing the former, he accepts the classification of layers introduced by the writer of this review, namely, in the visuo-sensory area: (1) superficial layer, (2) pyramidal layer, (3*a*) outer granules, (3*b*) line of Gennari, (3*c*) inner granules, (4) inner line of Baillarger, and (5) polymorph layer; and in the visuo-psychic region, (1) superficial layer, (2) pyramidal layer, (3) granule layer, (4) inner line of Baillarger, and (5) polymorph layer.

The exact visuo-sensory area of the Macacus is described and figured by the author, and he then refers to the published work on the visuo-sensory and visuo-psychic regions in the anthropoid apes and man.

He finally sums up the subject as follows: "Contrasting, then, the microscopic structure of the visual cortex of the primates with the lower mammals, we find that the main differences are the existence of an occipital *lobe*, the cortex of which consists of two structurally different regions: (1) visuo-sensory, which attains its maximum extent and complexity of cell and fibre structure in the ape, characterised by an increase of the width of the line of Gennari, a double layer of granules, and above the outer a deep layer of small and medium-sized pyramids; (2) visuo-psychic or associational, either not definite or as a very narrow zone in animals below the apes, and characterised by an increased depth of the pyramidal layer, owing to the presence of a third layer of large associational pyramids. This associational zone increases in area with the progressive evolution of the primates, and occupies a large part of the superficies of the occipital lobes in the highest types of human beings." . . . "My observations on the visual cortex of mammals thus tend to show a parallelism between the progressive development of the pyramidal layer in the visual

cortex of mammals and the progressive development of binocular vision."

Section III. deals with "the reciprocal simultaneity in the development of the visual directive and tactile motor executive faculties." The theory of binocular stereoscopic vision is discussed, and the author in this connection points out that "no true ideas of distance, form, extension, solidity and qualities of bodies generally would be conveyed to the mind unless they could be explored by the executive, locomotive, and tactile faculties." He concludes that "every visual perception becomes a complexus produced by an action on the retinæ propagated to the visual cortex, and a reaction of the whole brain based on the associative memory of past experiences."

In Section IV. the author deals with "the progressive development in the primates of the higher association centres; the effects on the visual cortex." He introduces diagrams to illustrate the diminishing degree to which the visuo-sensory area extends to the external surface of the hemisphere in the Macacus, the Chimpanzee, a Congo negro, and the ordinary European respectively. Dr Mott then remarks on the fact, proved by Elliot Smith, that the human visuo-sensory area always occupies the same superficies, and points out that "the placement of it depends upon its relative degree of displacement by cortex of later evolutional development and functional acquirement." The cause of this displacement to the internal aspect of the occipital lobe is considered by the author to be the striking, and to some extent variable, development of the parietal lobe in man. This point is illustrated by two hemispheres of the same brain. In the left the visuo-sensory area extends to the external surface, and the parietal development is poor. In the right the parietal lobe is more highly developed, and the visuo-sensory area does not extend to the lateral surface.

Dr Mott, curiously enough, has inadvertently illustrated his point in diagrams D and E, Fig. 27 (after Campbell), the originals of which were published by the writer of this review. The visuo-sensory area in this case extends to an unusual degree on the external surface, and the patient was a blind degenerate of poor cortical development, who died at the age of twenty-two years, after five years' residence in an asylum.

The conclusion of the author, from an examination of over 100 brains, is that an extension of the visuo-sensory area to the external aspect "of the occipital lobe on one or both sides is associated with a brain below the average in weight and convolutional pattern, such as occurs so frequently in congenital aments."

Section V. deals with experimental and clinico-anatomical

observations on the functions of the two histologically, phylogenetically, and ontogenetically different regions of the occipital lobe (*i.e.* visuo-sensory and visuo-psychic regions).

The functions of the visuo-sensory area are first discussed, and Dr Mott accepts the view of Henschen that the lower half of the area corresponds with the lower quadrants of the homonymous halves of the two retinæ, and the upper to the upper. He then discusses at length the question of macular representation, with especial reference to the views of Von Monakow and Henschen, and, after referring to certain of the more important published clinical cases of hemianopsy, he concludes that "the macular localisation, however, by clinical observation, is not yet satisfactorily determined."

The subject of the visual area is then considered from the experimental aspect, and the significance of the frontal eye-centres is discussed from the point of view of the development of the visual directive and the tactile motor executive faculties. In this connection Dr Mott suggests that the inexcitable frontal region in the neighbourhood of these centres may "consist of a zone of cortex which serves for the fusion of visual, equipoisal sensations, and those coming from the head and neck, trunk, and specially lower limbs."

With a view of functionally distinguishing between the visuo-sensory and visuo-psychic regions, the author refers to cases in which hemianopsy has occurred without loss of visual memory pictures, and in which the latter has existed in the absence of hemianopsy; and also to cases where both these deprivations existed. "It follows, therefore, that localisation of the two functions are probably in adjacent areas." Dr Mott here draws attention to the relatively large size of the pyramidal or latest-developed cells of the visuo-psychic region of the human brain. As a contrast to the large size of these late-developed human pyramidal cells, illustrations of the solitary cells of Meynert in the different orders of mammals are inserted. The drawings demonstrate that these cells, which are phylogenetically and ontogenetically of earlier development than the pyramidal, and which are in connection with and preside over lower neurones of practically similar functional value in all the higher mammalia, are of approximately the same size in all these animals.

Finally, in Section VI. the author refers to the researches of Bethe and Cajal on nerve cell fibrils, and obtains from these a probable explanation of the practically constant size of the motor cells of the visual cortex in different animals.

The paper is worthy of the reputation of its author, and, as a whole, is beyond the reach of criticism.

It may, however, be remarked that whilst Dr Mott lays especial

stress on the parallelism between the progressive development of the pyramidal layer in the visual cortex of mammals and the evolution of binocular stereoscopic vision, a similar and equally marked parallelism exists in the case of the latter, and in the progressive development of the middle cell or granule layer from a thin layer of granules to, in the primates, a thick double granule layer enclosing the line of Gennari. In the opinion of the writer of this review the latter probably bears a closer relationship to the evolution of binocular stereoscopic vision than does the progressive development of the pyramidal layer. In cases of congenital or long-standing blindness the line of Gennari is reduced in depth to the extent of 50 per cent., and the outer layer of granules by 10 per cent.; whereas the remaining layers of the cortex are unaffected in depth by blindness, and the cortex of the visuo-psychic region is entirely unchanged in depth. Further, the researches of Watson on the mammalian cortex, and of the writer of this review on the human cortex, in normal, under-developed, and demented cases, suggest that the degree of development of the pyramidal or outer cell layer is in the lower mammalia associated with the degree of educability possessed by the different animals, and that this layer is in man concerned with psychic or associational functions, these being pre-eminent in the prefrontal region, less marked in the visuo-psychic region, and least marked in the visuo-sensory area; in other words, that the pyramidal layer is phylogenetically and ontogenetically associational in function.

It is thus likely that the more purely sensori-motor functions which are concerned with binocular stereoscopic vision may have their *visuo*-sensory basis in the compound granule and Gennari layer of the area striata, whilst, in agreement with Dr Mott, such psychic phenomena as the formation of visual memory pictures would occur in the pyramidal layer of the visuo-psychic area. Under these circumstances the pyramidal layer of the visuo-sensory area, which is developed almost to the adult depth even in a case of anophthalmos of the age of one month, and which invariably develops earlier than the corresponding layer in the visuo-psychic region, would be concerned with the elaboration of the more elementary psychic products arising from visual sensation.

J. S. BOLTON.

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Review
of
Neurology and Psychiatry

Original Articles

PSYCHO-EPILEPSY.

By Sir WILLIAM R. GOWERS, M.D., F.R.S.

A PECULIAR feeling of fear, often intense, is sometimes the momentary aura of an epileptic fit. Followed by brief loss of consciousness, it may constitute minor attacks in the same patient. It is not met with in absolute isolation in ordinary epilepsy, as it was in those I am about to mention. In other patients an isolated sense of fear is replaced by an intense feeling of distress, with no other symptom. In both forms the subjective emotion begins and ends suddenly; sometimes its duration is longer than it is in cases of definite epilepsy.

A woman of 63, whose mother and brother had suffered from depression, sought advice for quite another malady—paralysis of the long extensor of the fingers of the right hand. It was probably due to hæmorrhage into the sheath of the nerve, since it was quite sudden in onset. But I learned also that for fifteen years she had suffered from attacks of the most intense depression, coming on quite suddenly, lasting for as long as five minutes, and ending as abruptly as they commenced. Sometimes, but not often, the ending was incomplete; it was a sudden, considerable diminution in degree, and a slighter, vague wretchedness continued for twenty-four hours. The transient attacks occurred once or twice a week, and were so brief that she

had come to take little notice of them. When they began, at the age of 49, she had persistent, trifling delusions for a few weeks, but these ceased and had never returned. The attacks of brief depression were never attended by nausea, vertigo, disturbance of the special senses, or any impairment of consciousness. Their peculiar character had continued unchanged during the long time they had occurred.

A case in which the sensation was that of fear was that of a man of 45 who had suffered for two years from attacks, lasting only a few seconds, of intense vague dread. He never lost consciousness, but the feeling of fear was such as to entirely absorb him, so that, for the moment, he scarcely knew where he was.

In a recent lecture (*Lancet*, June 8, 1907) on what I have termed "Vagal Attacks," because the symptoms are chiefly related to the functions of the pneumogastric nerve, I described some cases that suggest that the symptoms of an epileptic fit may sometimes be extended. They are protracted, as it were, and the features of the attack thereby changed much in character. This seems the only explanation of some attacks suffered by a man of 32, who was certainly the subject of minor epilepsy. For two years he had been liable to attacks of sudden unconsciousness, lasting, he thought, for ten minutes. Usually quite sudden, the loss of consciousness was sometimes preceded by a peculiar mental state, "thoughts passing through the mind like dreams." This was not momentary, like an ordinary psychical aura, but prolonged, lasting sometimes for three-quarters of an hour, until suddenly ended by loss of consciousness. This terminal loss was momentary; not prolonged for five minutes, as was that which occurred alone. Lying down, or putting his head low, occasionally prevented the loss, and the mental state then gradually subsided without other symptom. Similar as his attacks were to epilepsy, they were permanently arrested by nitroglycerine and strychnine. In the lecture referred to I have mentioned a case of similar character in which a prolonged peculiar brain state was suddenly terminated by a moment's loss of consciousness.

In the last case the psychical condition was mental rather than emotional. So it was in another patient, whose recurring symptom had much less resemblance to epilepsy, although its

relation to the latter was clear. The state was that of mental inertia, a condition often met with as a part of a "vagal" attack (*loc. cit.*) He was a solicitor, aged 28, whose uncle was epileptic. When 22 he began to suffer from giddiness, very brief, apparently petit mal. It occurred two or three times a year, independent of circumstances, whether walking or sitting still. Objects seemed moving to the left, and if walking, he himself had a tendency to deviate in that direction. If reading, the seeming motion of the letters towards the left made the type illegible. It endured only for a moment; then he was well. He never lost consciousness or fell. But for two years he had suffered also from the attacks that specially concern us. They were quite distinct in character and occurrence from the others, and he had one about once a month. Normally his mental power was acute, but quite suddenly his mind became inactive and could not be roused to work. Without any warning or special symptom, he found himself unable to follow any argument or train of thought, or to concentrate his mind on any special subject. He was quite unable to continue work. This state came on irrespective of fatigue, and generally without any special preceding mental exertion. Whatever the time of onset, the state went on through the rest of the day. The night's sleep always renewed his normal condition, which continued until another attack occurred.

This case suggests a question we cannot answer. Can this prolonged mental inertia represent a condition of the brain which, compressed into a moment, would have involved loss of consciousness?

REMARKS ON MYOCLONUS-EPILEPSY, WITH REPORT OF A CASE.[1]

By L. PIERCE CLARK, M.D., New York,

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THE case of myoclonus-epilepsy which I desire to report is of the family type, and is briefly as follows. The patient, a woman, aged 52, single, was admitted to the Craig Colony for Epileptics, September 30, 1904. Through Dr W. T. Shanahan's courtesy I am permitted to present many details of verified observation in the case. The paternal grandmother always suffered from headaches. A paternal uncle had three children, one boy and two girls, all of whom suffered from myoclonus-epilepsy similar to that of our patient. The disease is known in the family as "St Vitus' dance and fits." Whether epilepsy or myoclonus developed first in the cousins is not certain. The oldest of the myoclonic-epileptic cousins (female) married, and had one child, who lived to an adult age and showed no signs of the disease or other nervous affection. The mother died of unknown disease at fifty years of age. The two male myoclonic-epileptic cousins died of unknown diseases at the age of twenty-five and thirty respectively. A maternal grand-uncle was insane, and had several children who were insane; one grandson (second cousin to our patient) died of paresis. Several maternal relatives had tuberculosis. The father and mother of our patient were first cousins. Both suffered from chronic rheumatism. The father is still living and is a fairly healthy man of seventy-five years of age.

Our patient was a strong child. She had an ordinary attack of typhoid fever at ten years of age. She began school at five years of age, and made the average progress of her grades until thirteen years of age, when she had a typical grand mal epileptic fit at night, without obvious cause. The myoclonus, which either immediately preceded or followed the fit, began in the arms first. Although it was more or less constant after its inception, its paroxysmal intensity at stated periods is certain. The bad days

[1] Read before the N.Y. Neurolog. Soc., Dec. 3, 1906.

FIG. 1.

of myoclonus often interfered with piano practice. It may be interesting to note that our patient saw her myoclonic cousins a few months prior to her own disease, and was much affected by the sight. A possibility of similar contamination led Unversicht to isolate several healthy children in his first family, but they nevertheless developed the disease promptly on puberty. The order of myoclonic extension in our patient is not remembered. The fits were rather severe in her fourteenth year. She had seven severe fits in one night. From fifteen to forty-two years of age the attacks were less frequent, averaging one a month. She now has eight attacks a year—four grand mal and four petit mal, equally divided between day and night attacks. An aura of a cramp sensation in the right wrist was invariably present formerly, but it is now absent. For several years after the onset of her association disease she had increasing vertigo and myoclonic movements for two or three weeks before the genuine fits occurred, after which she was perfectly free from myoclonus for two or three days. Myoclonus never interferes with, nor does it occur during sleeep. Bromide and chloral, particularly the latter, improved her condition considerably during the thirteen years of their use, from the onset of the disease. She has taken no medicine for several years now. She is a Christian scientist. Coffee still has some quieting effects on the myoclonic movements. Patient had nocturnal enuresis until she was thirty-one years of age. It stopped without treatment. Involuntary urination never occurs during the myoclonic or epileptic attacks.

The epileptic fits are so classic in detail that they do not deserve mention. They have produced little or no mental enfeeblement. Some myoclonus is now present every day. The movements range in degree from slight fibrillary twitchings in individual muscles (myokimie), to that of universal involvement in all voluntary muscles (bad days). Extremes of heat and cold, mental stress and emotion excite the myoclonus.

At first the myoclonus often interfered with swallowing. Occasionally now, at the height of paroxysmal myoclonic days, foods and fluids are forcibly ejected at meals. She has broken her nose and received many disfigurements about the face by injuries due to falls in her myoclonic attacks (see Fig. 1). At the present report the myoclonic movements may be observed to rapidly succeed each other in a series of brisk, shock-like

muscular contractions, typical of myoclonus. All the muscles of the trunk and extremities are involved in this play of spasm movement, bewildering to describe. The large proximal muscles of the extremities are most involved, together with those of the trunk proper. The movements of the muscles of the two sides are a little asynchronous, the body and head are hurled to the right frequently at the end of the complex movement. In the extreme paroxysm all the muscles of the face are markedly affected, giving no expressive value to the features that can be described. The diaphragm and respiratory muscles are commonly involved. Aside from the ordinary checker-board of good and bad days, there are long periods of several days' duration in which a sort of status myoclonus intervenes. If standing, she is thrown from her feet by the shock-like contractions; if she remains in bed, she is almost hurled from it by the violent contractions. These periods are not often immediately followed by epileptic fits. There is never loss of consciousuess during any myoclonic movements. Speech is commonly interrupted thereby by the violence of respiratory involvement, but continuity of thought is never broken. The great flexors of the trunk are most involved, the contractions of the rectus abdominis are often painful. For several days following these severest periods of myoclonus a feeling of general exhaustion and muscle soreness exists. The knee-jerks are markedly diminished during the status myoclonus. The urine usually shows an increase of indican, but this finding is not constant. The nervous system shows no other abnormality, nor is there any hysterical stigma. The patient's writing often shows the myoclonic rockets and somersaults typical of myoclonus-epilepsy, even though the patient grasp the thumb of the right hand in the left hand to prevent wayward movements.

Myoclonus-epilepsy is still a rare disease. Even a keener differential knowledge of the motor disorders in epilepsy will probably not make it otherwise. But 108 authentic cases have been reported in about twenty years. My first and second papers in 1899[1] and 1902,[2] which established the association

[1] "Paromyoclonus multiplex associated with epilepsy." *Archives of Neurology and Psychopathology*, vol. ii., 1899.

[2] "The nature and pathology of myoclonus-epilepsy." *American Journal of Insanity*, vol. lix. No. 2, 1902.

disease as a clinical entity (and gave it the name of myoclonus-epilepsy), placed but four cases of my own observation on record out of a total of several thousand epileptics observed; while Mist of Kiel, who has gathered all the material on the subject from the many thousand epileptics passing through the great institution at Bielefeld during its half century of existence, reports but eight cases. Its statistical frequency can hardly be more than one to two or three thousand ordinary epileptics. Many writers have been in error in regarding Unversicht as the pioneer worker of the association disease. Unversicht wrote upon *family myoclonias*, and only incidentally mentions that many of his family type had epilepsy. Error has crept in also regarding the clinical manifestations of the disease. In myoclonus-epilepsy not only is the epilepsy somewhat atypical in its course and termination, as has been recognised in most reports, but the myoclonic symptoms almost invariably differ from the simple myoclonus of Fredrichs and others. In these the myoclonic spasm not only involves whole muscles of the trunk and the extremities in many cases, but also may be seen in the face and small muscles of the hands and feet.

Not a few of these reports have caused some writers to doubt the correctness of a diagnosis of myoclonus-epilepsy. Thus Bechterew and Koshewenikow diagnosticate such cases choreic-epilepsy, and others, especially the French writers, as multiple tics. The extreme rarity of chorea in epilepsy and the very unlike spasm in the tics should settle these doubts. Although the essentials of the myoclonias in the association disease are usually held to be a bilateral, clonic, lightning-like muscular spasm involving the trunk and proximal parts of the extremities, mostly or exclusively, cases are on record in which one or more of these so-called essentials are much modified or wanting altogether. It is interesting to note that in France, where the tics have had their most thorough exposition, the name "multiple tics" and Henoch-Bergeron's disease still do service for many obviously well-marked cases of simple myoclonus. Even in this country the term myokimie (Schultze), a form of fibrillary myoclonus, as well as multiple tics and chronic chorea, are frequent designations for myoclonus. In Russia also Koschewenikow's epilepsy can hardly be other than myoclonus-epilepsy. Not a few of Bechterew's many cases of choreic-epilepsy are

excellent illustrations of the association disease under consideration here. Again, choreic epilepsy is an extremely rare association in the experience of most neurologists. It would require no great distortion of the clinical picture of myoclonus-epilepsy to classify all of these peculiar grand tics and chronic choreas found associated with epilepsy as myoclonus-epilepsy.

While it seems desirable to include some irregular types of motor disorders associated with epilepsy under the general head of myoclonus-epilepsy, there is not a little tendency to class many cases of irregular manifestations of ordinary epilepsy itself in the association disease. For instance, it is not uncommon to see some initial spasm of epilepsy, the so-called motor aura, which causes the patient to execute some sudden flexion or extension movement of the extremities resembling a myoclonus spasm. Cases presenting these pseudo-myoclonic movements are often designated myoclonus, whereas they are but motor manifestations of ordinary Jacksonian epilepsy. These pseudo-myoclonic spasms seen in the initial phase of true epilepsy are always attended by a disorder or loss of consciousness. They are never multiple or quickly repeated, they affect the upper extremities only, are always bilaterally synchronous. They consist either in flexion of the segments of the arms or abduction of the entire upper extremity, producing a movement of the arms as though the patient were making an earnest attempt to maintain equilibration. The idio-muscular tremors occasionally seen in certain exhaustive phases of epilepsy proper are rhythmical and not modified by the will, and moreover, are never severe enough to produce locomotive effort, much less throw the patient to the ground as in myoclonus-epilepsy. Many cases of idio-muscular tremors are mistaken for Brissaud's chorea of the polymorphic type, which has a close analogy to Schultze's myokimie. During the past two or three years a few facial tics in epilepsy have been diagnosed myoclonus. The various facial tics seen in degenerative epilepsy have their maximum of expression in the face; they are not co-ordinate, and usually produce voluntary or reflex emotional acts, whose general characteristics remain the same, although subject to great periodic alteration in range and intensity. They are greatly under the dominance of the will. Coprolalia, echolalia,

achemesis and fixed ideas, impulsive and compulsive, are often associated with tics.

It is fairly well recognised to-day that the myoclonus-epilepsy develops on a degenerative basis, as that of epilepsy proper. There are no known instances of permanent recovery from the disease. Often the myoclonic symptoms improve for a time or mask themselves under more frequent and severe epileptic attacks. The same statement also holds true for the epileptic seizures. Spontaneous improvement for any great length of time in both classes of symptoms at the same time is rare, even under most favourable treatment.

It is quite generally admitted that the association disease is a cortical affection. The exact nature of the cortical degeneration is not clear, although Prout's analysis of my one case did not show a very dissimilar lesion from that of epilepsy proper. The destruction of the intranuclear network of cells of both sensory and motor type, or of the second and third layer type, demonstrated by Prout, has not been confirmed or denied. We know epilepsy is a cortical disease. The frequent interchange of symptoms of myoclonic for epileptic attacks, and vice versa, is one of the strongest reasons for placing the pathology of myoclonus-epilepsy in the cortex.

Bechterew's experimental production of myoclonic spasm by irritation of the Rolandic zone through a trephine opening in the skull is pertinent. He was able to produce myoclonic spasm entirely analogous to that which preceded the patient's epileptic attacks. Lundborg believes that in the highest type or most severe form of myoclonus, epilepsy supervenes. That myoclonus leads to epilepsy is interesting, but meets with many clinical objections in case-study of simple myoclonus and the association disease. There is little doubt, however, that an unstable nervous system and heredity are insufficient to account for the full development of the disease. From the probable pathology of myoclonus-epilepsy being in the cortex, one is led to think the pathogenesis of the disorder is closely bound up with that of epilepsy proper. In looking for a possible toxic or autotoxic cause for the disease there is a great diversity of opinion. In a review of the possible toxic agents Ferrero found one case of simple myoclonus that appeared to depend on a state of uric acid diathesis, while Landamy found essential myoclonus

in alcoholics, and Labbé, Grasset, and Ambland in chronic morphinism. The uniform absence and presence of indicanuria in certain cases of essential epilepsy, in myoclonus and in the association disease have been reported and consequently are of little value. Vanlair and Turtschminikow, in an endeavour to find the causative intoxicants of essential myoclonus, produced myoclonic contractions experimentally in animals by peripheral injections of carbolic acid. They believe that the drug caused an irritation of the sensory cells of the cortex through their peripheral endings, or in other words, that the myoclonus was a disturbance largely of the sensory motor reflex of the cortex. Wagner observed that myoclonic contractions followed extirpation of the thyroid, and has suggested a replacement therapy of thyroidin, which, it may be unnecessary to say, has been of no use.

Inasmuch as the true development of the disease occurs usually in the developmental period, exogenous toxins may be largely dismissed from consideration. Myoclonus-epilepsy usually develops during those years in which the thymus is undergoing involution. Therefore Lundborg thinks that it is reasonable to suppose the adult thymus may retard the appearance of the malady, and that when the functional activity ceases some other organ ought to assume its office. Lundborg as well as Wagner have sought to involve the thyroid and parathyroid in this connection. It is unnecessary to say that as yet practical results have not been forthcoming from these ingenious hypotheses. The motor disorders closely resembling myoclonus-epilepsy seen after extirpation of the thyroid and parathyroid in Jeandelise's experiments are highly instructive. The disorder was, however, much more acute, and moreover was not invariably present. In some experiments there was a general myoclonus amounting to tetanus. The motor disorder also showed intermittence. There seems to be a consensus of opinion as to the sequence of development of the motor disorder following these extirpation experiments; thus the motor disturbance begins as tremor-fibrillary twitchings, increasing to clonus, becoming more and more generalised and culminating in either an epileptoid or tetantoid state, which in turn is followed by an interval of rest, only to be repeated again and again until, as a rule, an unfavourable termination results. The terminal state in these experi-

mentally induced myoclonias is therefore a sort of status of myoclonus, as in one of my cases of myoclonus-epilepsy. The relationship of the thyroid and parathyroid to epilepsy proper and puerperal eclampsia furnishes much data for further speculation. The accumulating data of parathyroid experimental therapy should be illuminating on all these subjects. The therapy of venesection, transfusion, and oxygen inhalation in severe periods (status) speaks for some sort of toxic agent. It is discouraging to say that parathyroid extracts, while not yet extensively used, have been of no signal benefit in myoclonus-epilepsy. It may even be that the technique of the preparation and administration of the remedy is greatly at fault. Finally, we may say that while the whole matter of the pathogenesis of myoclonus-epilepsy is as far from solution as that of epilepsy proper, we are steadily annihilating the possible sources of pathogenesis, and the most feasible source of the toxin seems to be from the parathyroid bodies.

A SIMPLIFIED TECHNIQUE FOR ACCURATE CELL ENUMERATION IN LUMBAR PUNCTURE.

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THE practical utility of many methods of clinical investigation is greatly dependent on the facility with which observations can be quantitatively estimated, and so the results of different workers compared. This is strikingly seen in the case of such methods as Widal's test, where an enormous accession of utility at once followed on the discovery of how to standardise the results; without this discovery the method might still have been to-day of only theoretic interest. Consequently, when a new method has been proved to be of great value, it behoves workers to try to find a means of so stating their results that they can be compared quantitatively with those obtained by other workers. The method of lumbar puncture is still in a state of incomplete evolution in this respect in spite of the extensive investigations that have been made by it. Of the valuable information that

it yields only some can be stated in quantitative terms. For instance, the pressure of the fluid can be accurately measured by the procedure described by Quincke (1); the amount of albumin can be determined by Reiss' process (2) or, if it is very small, by the more delicate one of Nissl (3). With the important question of the cell content of the fluid, however, only the roughest methods of cell estimation are in general use, and the object of this paper is to consider whether we have not at our disposal the means of making this estimation in a more accurate and ready manner.

The technique for the cytological study of the cerebro-spinal fluid is usually carried out according to the lines laid down in the classic writings of Widal, Sicard, and Ravault. The three cubic centimetres withdrawn are centrifugalised for ten minutes in an instrument that revolves 3000 times per minute, the supernatant fluid is completely decanted and a film is made from the deposit. The film is fixed with alcohol-ether, stained by Ehrlich's or any other suitable stain, and examined under an oil-immersion lens. By this means the relative proportion of the various cells present can be accurately determined by a differential count, which, of course, is of the greatest service in making a diagnosis. The absolute number of cells can, however, be only roughly indicated thus, and the estimation of it is largely a matter of individual judgment. When a great number of cells is present in each field, the existence of an abnormal leucocytosis, or lymphocytosis, as the case may be, is obvious, but when this number is small it is frequently very difficult to say whether it is of pathological significance or not. Personal experience is here of great importance, but even then it is impossible to give any data that can accurately be used for comparison. Ravault (4) has suggested the following convention: he calls it a "pronounced" reaction when more than twenty cells are present in an oil-immersion field (exact size not stated), a "moderate" reaction when seven to twenty cells are present, a "mild" reaction when four, five, or six are present, and a "negative" reaction when there are less than four. Although this convention has proved of some service in default of a more accurate procedure, it is clearly only a rough makeshift and is open to many criticisms. Nissl (5) has indicated the fallacies implicit in the procedure, and has formulated five

precise objections to the use of such a method as a quantitative observation. He concludes that, even with the most careful technique, it may occasionally be possible to find eight or ten cells in a field without any pathological significance attaching thereto.

The only serious advance on this technique was that made by Laignel-Lavastine (6), who suggested that after centrifugalisation only some of the fluid be decanted, leaving enough to make an emulsion with the deposit. He pipettes this emulsion into a Manassez chamber and estimates the number of cells per cubic millimetre. Then the number of cells per cubic millimetre of the original fluid is clearly $\frac{N \times D}{V}$, where N is the number of cells per cubic millimetre in the emulsion, D the quantity (in cubic millimetres) of fluid and deposit left to make the emulsion, and V the total amount (in cubic millimetres) of the original fluid. De Buck (7) has also employed this procedure, but he uses a Thoma-Zeiss chamber with a modification of the Prague method of counting.

The theory of the procedure is therefore quite straightforward: the fluid is concentrated till all the cells are in a conveniently dense solution, the percentage of them in this solution is estimated, and the percentage in the original fluid calculated by knowing the relation of the solution examined to the original fluid. In practice, however, the procedure is more cumbrous than might appear, which probably accounts for its being rarely adopted. The main reason for this is that the actual number of cells is much smaller than that present in blood, even when this is diluted to the usual extent, so that the enumeration is both tedious and fallacious. For instance, De Buck counts the cells present on 256 squares of an Abbé-Zeiss slide, that is in about a fifteenth of a cubic millimetre; in a case of tabes or general paralysis, even if the fluid is concentrated by centrifugalisation, the number of cells in the emulsion examined is frequently less than twenty in this amount, so that the possible error is very considerable (over 30 per cent.).

It thus becomes specially advantageous to adopt some of the more modern methods of using the Thoma-Zeiss chamber by means of which large areas can be rapidly inspected. The best of these is the "field" method, first described by Stengel (8).

In this the squares marked on the slide are ignored, and the number of cells in each field of vision of the microscope counted, a larger unit being thus obtained. As usually employed this method entails a rather elaborate calculation, which, however, can be avoided in a way I shall presently indicate. The area of the field must of necessity be calculated by the formula πr^2, where r is the radius and π a constant, $3{\cdot}1\dot{4}2857\dot{1}$. The radius can most accurately be determined by adjusting the slide and the magnification of the microscope till a square formed by the ruled lines—and containing several little squares—touches at its four corners the circumference of the field; the radius is then equal to the length of one side of this square divided by $\sqrt{2}$ (corollary to Euclid, Book i., Prop. 47). The length of the line in question, and of similar lines, is easily found, because it is made up of a number of marked sections, each of which constitutes the side of a little square and measures one-twentieth of a millimetre. An easier though rather less accurate way of finding the length of the radius is by moving the Abbé-Zeiss slide until one of its ruled lines coincides with the maximum diameter of the field. The line is measured as just indicated and of course the radius is half its length.

The field method is commonly employed in the following way. Recognising the fact that the diameter, and therefore the area, of the field can be varied at will by adjusting the magnifying powers of the microscope, a diameter is chosen which contains an integral number of the marked sections measuring each one-twentieth of a millimetre. The cubic capacity of the underlying space is then calculated as above, and its relation to the standard, a cubic millimetre, thus ascertained. This relation is usually a complicated fraction. The average number of cells per field is then found by counting a number of fields, and the result is divided by the above fraction, and multiplied by the degree of dilution of the original fluid. In the case of the cerebro-spinal fluid the number would have to be multiplied by the Laignel-Lavastine formula given above.

It will thus be seen that the process is quite cumbersome, and the trouble taken is disproportionate to the accuracy of the results, on account of the error due to the smallness of the number of cells. It has occurred to me, however, that the process might be greatly shortened by the following device,

which also adds considerably to the accuracy of the results. Instead of choosing a diameter which contains an integral number of marked sections, let one be chosen in such a way that the cubic capacity of the space constitutes a simple factor of a cubic millimetre. For instance, if this space occupies exactly $\frac{1}{100}$ of a cubic millimetre, evidently counting the cells in 100 fields gives us the number of cells per cubic millimetre without any further calculation whatever. All that then remains to do is to calculate the number per cubic millimetre of the original fluid. The only preliminary work that has to be done is the adjustment of the magnification until the diameter of the field is of the requisite length. This takes only a minute or two, and, once it is done with a given microscope, the knowledge is gained for good that the use of such and such lenses gives a field of the required dimensions, so that the diameter need never be again measured. The diameter I recommended when I described the method in connection with hæmatological work (9) was equal to $6\frac{2}{3}$ of the marked sections; a field of that size is obtained with the magnification of Leitz Oc. 1 and Obj. 7 lenses, the draw-tube—in a Leitz II. A microscope—being at 170, and the space thus obtained is $\frac{1}{125}$ of a cubic millimetre. In this case the counting of the leucocytes in twenty-five fields gives, provided the dilution is 1 in 20, the exact number of leucocytes per cubic millimetre merely by the addition of two terminal ciphers.

The next problem is to decide what size field will be the most convenient to employ in the case of cerebro-spinal fluid. This decision is arrived at only as the result of practical experience, and I would suggest the following technique as being the most advantageous.

We may divide the cases in which a cytological study is necessary into two fairly defined groups: those with a great excess of cells, causing obvious turbidity of the fluid, such as is often seen in cases of meningitis, and those with a much smaller number, such as is seen typically in tabes or dementia paralytica.

In the former group the number of leucocytes per cubic millimetre is frequently as great as several hundred, in other words as great as that in normal blood when diluted twenty times, as it usually is in the "white" pipette of the Thoma-Zeiss hæmocytometer. In this case previous centrifugalisation is unnecessary, and it is sufficient directly to count the cells present

in half a cubic millimetre, and then simply to multiply the result by two. The readiest means of doing this is to choose a field corresponding to a cubic space of $\frac{1}{80}$ of a cubic millimetre. This is found to be so when the diameter of the field covers eight sections (sides of little squares). All that need be done then is to count the cells present in twenty of these fields, and repeat the count in a second drop. We then know the number of cells present in half a cubic millimetre. This procedure is equivalent to counting the cells present in 2000 little squares, and is thus far more accurate than that employed by De Buck, who counts the cells in only 256 squares, though it occupies very little more time. If the turbidity of the fluid is specially pronounced, it is sufficient to count the cells in twenty fields, using one drop only, and then to multiply the total by four, which gives us the number per cubic millimetre.

In the second group Laignel-Lavastine's manœuvre has to be adopted in order to obtain a sufficiently great number of cells ; this is important, for the accuracy of the results depends on, among other factors, the number of cells counted. The readiest way of carrying out the manœuvre is to employ a centrifugalisation tube that has the lowest centimetre graduated in divisions of tenths. Then enough liquid is retained after centrifugalisation and decantation to form an emulsion with the deposit, and the exact relation of the liquid retained to the original quantity is obvious at a glance. Even then the number of cells obtained may be so small that special manœuvres have to be adopted. For instance, out of thirty-five cases of G.P.I. that presented a meningeal reaction, Laignel-Lavastine (10) found that in twenty-eight cases the number of cells per cubic millimetre was under ten. When this is so, greater accuracy is obtained by counting the cells in three separate drops, thus ensuring a more uniform distribution. In doing this it is convenient to employ a field corresponding to $\frac{1}{90}$ of a cubic millimetre and to count the cells in thirty fields in each of the three drops. This is equivalent to counting the cells in four thousand squares, more than fifteen times the number recommended by De Buck for the same purpose. A field of this size is obtained by using a diameter containing 7·5 sections.[1]

[1] The odd half, like most of such fractions, can be accurately measured by the following device. It will be remembered that on an Abbé-Zeiss slide every fifth section is divided by a line the function of which is, in conjunction with three other similar

An actual example, from an advanced case of tabes, will best demonstrate the simplicity and accuracy of their procedure. Three cubic centimetres of cerebro-spinal fluid were withdrawn and centrifugalised. A slight deposit was then visible and the supernatant fluid was decanted only until the level of the remainder stood at the sixth division; the remainder was thus one-fifth of the original quantity. The deposit and small amount of fluid were thoroughly stirred by rotating the tube rapidly and evenly. A drop was pipetted with a platinum loop on to each of three Abbé-Zeiss slides, covered and allowed to settle. The microscope magnification was adjusted until seven and a half little sections formed the diameter of the field, a procedure that takes a minute or two. The cells were counted in thirty fields and this was repeated with the other two drops. The total number of cells in the ninety fields was forty-three, which therefore was the number per cubic millimetre of the emulsion. Consequently the average number of cells per cubic millimetre of the original cerebro-spinal fluid was 8·6. The whole time occupied, including ten minutes for centrifugalisation, was twenty-five minutes and in the same time a film was made for microscopic examination. It will be observed that there was no step in calculation that could not be instantaneously performed mentally.

A few further remarks might be made on some points in technique. When the fluid is quite turbid centrifugalisation is not necessary, except of course for film purposes. When however there are clinical grounds to suspect that the number of cells will be small the fluid must not only be centrifugalised but this must be done thoroughly, preferably for ten to fifteen minutes. Nissl and Devault (11) have demonstrated that after centrifugalisation for only a couple of minutes, even with an instrument that rotates 3000 times a minute, there may be an appreciable number of cells still in the supernatant fluid. Again, when the emulsion containing all the cells is obtained it

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lines, to enclose sets of sixteen little squares. If now the end of the line that is to be measured is placed at the right-hand corner of a little square, so that the line from right to left traces first the sides of two unbisected squares, then that of a bisected square, and lastly those of four unbisected squares, we have seven sections measured, and the remaining half is already marked off by the vertical line that bisects the square that comes next in order to the left.

is important to mix this thoroughly so that the drops taken shall be representative of the whole.

The presence of blood is sometimes a practical source of embarrassment. If this is due to the puncture of a small vein clear fluid can generally be obtained by discarding the first portion that flows. Blood that is originally present in the cerebro-spinal fluid is frequently laked, so that only shadow erythrocytes are visible. It is sometimes difficult to distinguish erythrocytes from lymphocytes in the stained film, whatever technique be used, as Nissl (12) has pointed out. In the process of making the total count the difficulty due to the presence of blood can be avoided by adding a minute quantity of 30 per cent. B. P. dilute acetic acid, care being taken to allow in the calculation for this dilution. The acid has the further advantage of making nucleated cells more easily visible.

There are also some practical matters to be attended to in the actual counting of the cells. The most important point here is to be sure that empty fields are fairly counted in with fields containing one or more cells. It is a matter of great difficulty, which is appreciated only by practical experience, to avoid the habit of unconsciously moving the field until the eye catches sight of something positive. It is unquestionably better to glance away every time that the slide is moved. I need hardly say that a mechanical stage is practically essential, if only to ensure that the same region is not inspected more than once. Another rather important matter is the question as to whether cells should be included when they are only partly in the field. Clearly in a case where the excess of cells is only slight two observers would get very different results if one counted every cell any part of which was in sight in the fields examined while the other counted only those cells that were entirely in the fields. Some time ago I suggested (13) that Lyon and Thoma's (14) rule relating to the inclusion of erythrocytes in the Abbé-Zeiss squares be adapted to the "field" method of counting cells in the following manner. Imagine a line forming a vertical diameter of the field parallel to the observer's sagittal suture and let all corpuscles seen to the left-hand side of this line be counted as being within the field, whether in fact they are wholly so or only partly; on the opposite right-hand side only the corpuscles which are wholly in the field should be

counted, whilst those touching the circumference should be ignored.

Finally, the pre-eminent importance of securing data adequate in amount cannot too often be insisted on. Especially is this so when it is a question of deciding whether the borderland between physiology and pathology has been crossed, as for instance in cases with a small number of cells in the cerebro-spinal fluid. Laignel-Lavastine (15) states that one should attach pathological significance only to liquids containing more than 0.5 cells per cubic millimetre. Even if a liquid containing 0.5 cells to a cubic millimetre is concentrated to one-tenth and 4000 Abbé-Zeiss squares be inspected, only five cells will be seen. Now the possible error due to paucity of data alone is here over 60 per cent., in other words the number really present might prove in a larger count to be anything between two and eight. If we accept twice this number, that is one cell per cubic millimetre of the original fluid, as being the lowest indicating pathological significance, then if this is the exact number of cells present its existence can be established with certainty only by counting the cells present in over eight million squares. Put in another way, by using ordinary procedures we can be sure of the existence of one cell per cubic millimetre only when there are really more than this number present. As a matter of fact if we count only the cells present in 4000 squares, that is in one cubic millimetre—a sufficiently extensive procedure for routine use—there must be actually present at least 1.5 cells per cubic millimetre before we are justified in stating that the number present exceeds the pathological limit of one per cubic millimetre.

I append a few instances bearing on this subject and also a summary of the procedures suggested, though from the principles indicated above it is easy to modify these according to the number of cells that may be anticipated in different cases.

Summary of Methods Suggested.

Method.	Diameter of Field.	Cubic Capacity corresponding to Field.	No. of Slides prepared.	No. of Fields examined on each Slide.	No. of Abbé-Zeiss Squares inspected.
I.	8 sections	$\frac{1}{80}$ c.mm.	2	20	2000
II.	7·5 „	$\frac{1}{100}$ „	3	30	4000

Examples illustrating the Above Methods.

Method.	Total No. of Cells in Fields examined.	No. of Cells per c.mm. of Emulsion.	Degree of Concentration.	No. of Cells per c.mm. of original Fluid.
I.	75	———	1 : 1	150
	90	———	1 : 1	180
II.	200	200	1 : 10	20
	75	75	1 : 5	15

Examples illustrating Influence of Extent of Data on Accuracy of Conclusions.

No. of Cells actually counted.	Possible Error due to Paucity of Data.
300	8 per cent.
200	10 „
100	14 „
75	16 „
50	20 „
25	28 „
20	31 „
15	37 „
10	45 „
8	50 „
5	63 „

Examples illustrating Data necessary to justify Statements as to Actual Number of Cells present.

	No. of Cells (average per c.mm. of original Fluid) the presence of which it is desired to confirm.	No. of Cells (average per c.mm. of original Fluid) the presence of which is necessary to justify the foregoing statement.					
Degree of Concentration of Fluid examined.		1 : 1		1 : 10		1 : 5	
No. of Squares inspected.		2000	4000	2000	4000	2000	4000
	1	6	4	2	1.6	2.4	2
	2	8	5.2	3.1	2.8	3.8	3.1
	5	12	9.4	6.6	6.1	8.8	6.6
	10	18.8	15.6	12.2	11.5	13.3	12.2
	20	31.2	27.8	23	22	24.5	23
	25	37.6	33.2	27.4	27.5	30	27.4
	50	66.4	61.2	55	53.5	57.2	55
	100	122.4	115.2	107	104.6	110	107
	200	230.4	221	209	206.5	214	209
	300	340	325.5	311.5	308	316	311.5

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THE SYMPTOMS DUE TO PERIPHERAL NEURITIS OR SPINAL LESIONS IN DIABETES MELLITUS.

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In many cases of diabetes mellitus, nervous symptoms occur which are due to changes in the peripheral nerves, or in the fibres of the posterior nerve roots just after they have entered the spinal cord. There are several clinical forms of these affections, and the following description is based on the cases which have come under my care.

1. In the most common form the patient complains of pains in the legs; usually they are of a dull, aching, or gnawing character, and they may be so severe as to prevent sleep. Cramps in the calf muscles are common. The muscles of the legs are tender on pressure. There may be no other symptoms or signs on examination of the nervous system.

2. In other cases cramps occur frequently in the calf muscles, and there is slight tenderness of these muscles. On the soles of the feet and on the big toe nails there is loss of the vibrating sensation, tested by a vibrating tuning-fork; but on the malleoli (external and internal), and on the tibiæ, and on other parts of the body the tuning-fork sensation is normal. All other forms of sensation are felt, and the tendo-Achillis-jerks and knee-jerks are present. [For testing the vibrating sensation a large tuning-fork is necessary. The foot of the tuning-fork may be placed on the sole of the boot, if it is not too thick; or it may be placed directly on the sole of the foot (at the heel and at the anterior

part), or on bony prominences. If a penny be placed on the sole of the foot and the foot of the vibrating tuning-fork held firmly against the penny, often the vibrating sensation is felt better.]

3. In more severe forms the chief symptoms are aching or gnawing pains in the legs and feet, hyperæsthesia, with burning sensation and tenderness in the toes, or all over the feet, or in the feet and legs. The affected parts may be so tender that the patient cannot bear the bed-clothes to touch them; also he may not be able to bear one leg on the top of the other. In some cases there is a sensation of numbness and tingling in the feet or legs. There is great tenderness of the muscles, especially the calf muscles. In addition to the gnawing, dull pains there may be "neuralgic" pains. Sensation to tactile impressions, to painful impressions, and to temperature is not impaired, but often there is loss of the vibrating sensation, tested by placing the foot of the vibrating tuning-fork on the big toe nails and on the soles of the feet (in the region of the heads of the metatarsal bones and at the heel). In many cases the vibrating sensation is also lost over the internal and external malleoli and over the tibia, whilst in other regions of the body this sensation is present at the parts where it is felt normally. When the pain and tenderness is more severe in one leg than the other, the vibrating sensation may be lost over a wider area in the more painful leg—sole of foot, big toe nail, malleoli, and tibia on the more painful side; sole of foot only on the other side. The tendo-Achillis-reflexes are usually lost, but the knee-jerks may be present. In other cases the knee-jerks are also lost. There is sometimes sweating and hyperæmia of the feet, and the tenderness in the soles may be so great that the patient is unable to walk.

4. In another group of cases the pain in the legs is slight, but the tendo-Achillis-reflexes are lost, whilst the knee-jerks are present; or both tendo-Achilles-reflex and knee-jerks are lost.

5. In the four forms just mentioned there is no paresis or paralysis; but in a rare group of cases there are paralytic symptoms in the legs like those of alcoholic neuritis—paresis or paralysis of the anterior tibial muscles, dropped feet, difficulty in walking, and high-stepping gait. The pain, tenderness, and muscular hyperæsthesia described in the other groups of cases are also present, in varying degree of intensity; cutaneous

sensation may be diminished or lost in the legs, as in alcoholic neuritis.

6. A few cases have been recorded (pseudo-tabes) in which there has been unsteadiness in walking, (?) ataxia, along with weakness of the leg, and the other symptoms already described in group 4.

7. In rare cases there are perforating ulcers on the feet, like those of tabes, with pains in the legs and loss of the tendo-Achillis-jerks, or of both Achillis-jerks and knee-jerks. Other symptoms already mentioned may or may not be present.

8. Nervous symptoms are occasionally present in other parts of the body. The pain and hyperæsthesia, with or without paresis of muscles, may be localised to the region of the distribution of the anterior crural nerve of the thigh, on one or both sides. In other rare cases the arms are affected, the symptoms being similar to those in group 3 (brachial neuritis). The neuritic symptoms have been occasionally localised to the sciatic nerve, or to a nerve of the arm.

Of all these forms, 1, 2, 3, and 4 are by far the most common; actual paresis or paralysis, as in group 5, is exceedingly rare.

The chief symptoms of these cases of so-called diabetic neuritis are therefore :—

(1) Subjective symptoms: Pain and burning sensation; tenderness and hyperæsthesia of skin and muscles.

(2) Loss of the vibrating sensation on the feet, or feet and legs.

(3) Loss of the tendo-Achillis-reflexes; the knee-jerks being present in some cases, lost in others.

In the majority of cases pain, tenderness, and hyperæsthesia are the prominent symptoms. As Leyden has pointed out, the neuritis of diabetes is usually of the hyperæsthetic form. Though there may or may not be a sensation of numbness and tingling, nevertheless on objective examination usually no loss of sensation to tactile impressions, pain, or temperature can be detected; and usually *the only objective sensory defect is loss of the vibrating sensation,* as tested by a vibrating tuning-fork placed with its foot on the soles of the feet, on the malleoli, and on the inner surface of the tibia.

True paresis or paralysis, and anæsthesia to tactile sensation, pain, and temperature are very rare.

The tendo-Achillis reflex is often lost in diabetes; in 50 consecutive cases of diabetes (chiefly private patients) I found the Achillis-jerks both lost in 19, present in 29, one absent and one present in 2.[1]

The knee-jerk is also often lost; in 100 cases of diabetes, chiefly hospital patients,[2] I found the knee-jerks both lost in 49, one lost and one present in 6, both present in 45. But in 50 private patients I found the knee-jerks both lost in 6 only; one lost, one present, in 1 case; both present in 43.

As in tabes, the Achillis-jerks are usually lost in diabetes before the knee-jerks. When the Achillis-jerks are lost the knee-jerks may be lost or present, but when the Achillis-jerks are present the knee-jerks are nearly always present. Thus, in 50 cases of diabetes already mentioned, the Achillis-jerks were both lost in 19, but the knee-jerks were lost in only 8 of these cases.

The vibrating sensation is lost in the legs (at some part where it is felt normally) in many cases of diabetes, although other nervous symptoms may be very slight.[3]

When there are perforating ulcers on the feet the Achillis-jerks may be lost and the knee-jerks present, or both reflexes may be lost.

Resemblance of the Symptoms to those of Tabes.

From the description just given, it will be evident that many symptoms which occur in tabes may be met with in the nervous complications of diabetes, as for example the loss of knee-jerks and of tendo-Achillis-reflexes, perforating ulcers, pains in the legs, impairment of sensation, and sexual impotence. In diabetes, as in tabes, the tendo-Achillis-reflex is usually lost before the knee-jerk; in both affections, when these two deep reflexes are lost, the abdominal and epigastric reflexes are very often increased. The pains in the legs in diabetes are usually of a dull, aching, or gnawing character, and the typical shooting pains of tabes are usually absent; but in a few cases which have come under my observation, the description of the pains, given by the diabetic patient, has corresponded to that of the

[1] *Rev. Neurol. and Psychiat.*, October 1903.
[2] *Lancet*, July 17, 1897.
[3] *Lancet*, April 1, 1905.

sharp, stabbing, momentary pains of tabes. In diabetes, as in tabes, very often the first objective sensory symptom is loss of sensation to the vibrating tuning-fork, whilst other forms of sensation have not been lost.

The diagnosis from true tabes is, however, usually very easy. The difficulty occurs chiefly in cases in which the thirst and diuresis have been absent or slight, and examination of the urine has not been made. The patient has often sought advice on account of pain in the legs, or on account of a perforating ulcer; examination has revealed loss of knee-jerks and of the tendo-Achillis reflexes, and a diagnosis of tabes has therefore seemed probable. Hence it is a good practical rule never to omit the examination of the urine for sugar in all cases of severe pains in the legs, in sciatica, in cases in which the knee-jerks or Achillis-reflexes are absent, and in cases in which the symptoms of multiple peripheral neuritis are present.

The urinary condition is usually diagnostic. There are, however, several points of difference as regards the nervous symptoms. The usual difference with respect to the pain has been already referred to. Hyperæsthesia of the muscles, tenderness of the calf muscles, is very common in the diabetic nervous complications, whilst this tenderness is usually absent in tabes; in fact, in tabes very often there is muscular analgesia. The deepest pressure of the calf muscles in tabes often fails to produce any pain, whilst in the diabetic nervous complications slight pressure usually causes great pain. Girdle sensations and zones of diminished cutaneous sensation round the chest or abdomen are common in tabes, but do not occur in diabetes. The Argyll-Robertson pupil, gastric and other visceral crises, Charcot's joint disease, bladder symptoms, muscular hypotonus (in absence of paresis) occur in tabes, but not in the diabetic nervous affections. Primary optic atrophy occurs in 10 to 15 per cent. of the cases of tabes. I have examined the optic discs of a very large number of cases of diabetes, but have never met with a single case of optic atrophy. I am inclined to think that in the few cases on record, in which optic atrophy has been found in diabetes, the atrophy has been due to some accidental complication.

Pathology.—The slight nervous symptoms may occur in any form of diabetes, but the more severe nervous symptoms are met

with chiefly in the chronic forms of diabetes, sometimes in the mild cases; and usually, but not always, the patients are over 40 years of age. The severe nervous complications are very rare in young patients suffering from an acute and severe form of the disease.

From the similarity of many of the symptoms to those of peripheral neuritis, due to "alcoholism" and other causes, the nervous symptoms in the limbs in diabetes have been attributed to peripheral neuritis. This is the cause of the severe nervous symptoms in many cases. In a number of cases pathological examination has revealed a degenerative or parenchymatous neuritis in the nerves of the limbs affected.[1] But in other cases in which there have been simply loss of knee-jerks and slight pains in the legs, I have not found any changes in the small peripheral nerves of the legs; a more thorough examination, however, of the *most minute* intra-muscular nerve fibres might possibly have revealed slight degeneration.

But it is possible that the loss of knee-jerks and Achillis-jerks and some of the nervous symptoms may, in certain cases, be due to degeneration in the spinal cord affecting the intra-medullary fibres of the posterior nerve roots; or at least these changes, as well as changes in the peripheral nerves, may be the cause of nervous symptoms.

In some cases of diabetes I have found changes in these intra-medullary fibres of the posterior nerve roots and in the posterior columns of the cord.[2] Similar changes in the posterior columns have been reported by others. These changes are probably due to the toxic condition of the diabetic blood.

The following is a summary of the spinal changes:—

In sections stained according to Marchi's method, numerous degenerated fibres, stained black, were seen in Goll's columns in the cervical region; and a few scattered degenerated fibres were seen both in Goll's and Burdach's columns in the dorsal and lumbar regions.

In cases which I have examined, degeneration, indicated by black dots, was also seen in the fibres of the posterior nerve roots in their intra-medullary course, especially between the posterior surface of the cord and the posterior horn of grey matter. The degeneration affected the median bundle of the

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[1] Fraser, T. R., and Bruce, A., *Edin. Med. Journ.*, 1896, p. 300; (review of other cases given in writer's book, "Diabetes Mellitus and its Treatment," Edin. 1898).

[2] See *British Medical Journal*, Jan. 16, 1904, and Feb. 24, 1894, and *Medical Chronicle*, Oct. 1905; "Diabetes Mellitus and its Treatment," Edin., 1898, p. 242.

posterior root fibres, and commenced directly these fibres had passed through the spinal pia-mater. The degeneration of the posterior root fibres was best seen in the lumbar and cervical regions. External to the cord (external to the pia-mater) the posterior roots were usually quite normal. (In a few sections there was degeneration of the posterior root fibres, but only for a very short distance, just outside the pia-mater.) Lissauer's zone was not affected. These changes in the posterior root fibres very closely resembled, and had the same localisation as the degeneration of the intra-medullary fibres of the posterior roots in early tabes and in certain cases of intra-cranial tumour.[1]

The changes in the posterior columns are evidently, in some cases, secondary to the degeneration of the intra-medullary fibres of the posterior nerve roots, *i.e.* they are of the nature of ascending degeneration. As already mentioned, sections stained according to Marchi's method revealed degenerated fibres in the posterior columns, which were most numerous in Goll's columns of the cervical region. Also in the columns of Goll in the cervical region, aniline blue black, van Gieson's stain, and Weigert's stain revealed slight increase of the neuroglia. The nerve fibres of Goll's columns were mostly diminished in size, but close to the posterior median septum some fibres presented swollen axis-cylinders and distended myelin sheaths.

In some specimens changes were well seen by the naked eye on transverse section of the cord hardened in Müller's fluid. The affected parts of the posterior columns of the cord were paler than the rest of the white matter. In the dorsal region both Burdach's and Goll's columns were affected (pale), whilst in the cervical region Goll's columns only were pale.

In these cases the microscopical changes were much less distinct than those seen by the naked eye.

Treatment.—Often the nervous symptoms here described can be greatly relieved by medical treatment. It is important to give a careful dietetic and medical treatment suitable to the form of diabetes from which the patient is suffering. For the relief of the pains I have found the following drugs very useful: aspirin, gr. 10 or 15 three or four times a day; sodium salicylate, gr. 10 or 15 three or four times a day; antipyrin, gr. 10 three times a day. I have found extract of belladonna and glycerine (equal parts) or cocaine ointment (B.P.), applied to the painful and tender parts, to be of service.

[1] *Medical Chronicle*, Oct. 1905.

Abstracts

ANATOMY.

THE MAMMALIAN CEREBRAL CORTEX, WITH SPECIAL
(266) **REFERENCE TO ITS COMPARATIVE HISTOLOGY. I. Order Insectivora.** GEORGE A. WATSON, *Archives of Neurology*, Vol. iii., 1907.

THIS paper contains a portion of the outcome of an investigation into the structure of the cortex cerebri of various mammals, an inquiry into the functional significance of the cortical layers forming the primary purpose of the research. The present part deals with the brains of the British Insectivores, the mole, shrew, and hedgehog, and is almost limited to an account of the neopallium from the point of view of its cell lamination. Only a few of the statements contained in the paper can be briefly summarised in this abstract.

The classification of the cortical layers adopted is that introduced on developmental grounds by Bolton. The latter considers that the human cortex is constructed on a general plan of five laminæ, viz.: (1) molecular, (2) pyramidal, (3) granular, (4) inner line of Baillarger, (5) polymorphic. Of these only three are primarily cell layers, *i.e.* 2, 3, and 5; layers 1 and 4 being primarily fibre layers, although containing nerve cells, *e.g.* the cells of Cajal in layer 1, and the Betz cells, or solitary cells of Meynert in layer 4. Great differences naturally exist in cell form, and in the depth of the several laminæ in different mammals and in different regions in the same mammal, owing either to sub-evolution of a layer, or to hypertrophy and specialisation of a layer or part thereof. When, however, this has occurred it is unnecessary to assume that the lamination in these mammals or in these regions is *essentially* different. The above classification is therefore believed to hold good as a primary basis in all regions of the cortex throughout the mammalian series. Owing to the relatively greater development of the cell constituents of layer 4 in certain lower mammals, and to the difficulty sometimes found in accurately separating layers 4 and 5, the writer refers to these layers together as "infra-granular" cortex, and to the true pyramidal layer as the "supra-granular" cell layer.

An attempt has been made to map out the neopallium into different regions from variations in cell lamination. The chief areas so delimited are: 1. Motor—in which comparatively large cells (probable homologues of Betz cells) occur in layer 4; this occupies

a considerable portion of the dorso-lateral and a small part of the mesial aspect—laterally it ends abruptly, but anteriorly, posteriorly and mesially its limits are not clearly definable. 2. General sensory—external to 1—a large field showing a well developed granular layer, hence thought to represent projection (sensory) cortex, and probably to possess kinæsthetic functions. Areas 1 and 2 are in every sense the best developed regions of the neopallium; they are readily distinguishable in the mole, less readily in the shrew, and with difficulty in the hedgehog. Surrounding these two fields, anteriorly, posteriorly (also mesially), and laterally are areas of undifferentiated cortex which show no very special features in their cell lamination. Mesially, the most prominent region surrounds roughly the fissura hippocampi and is termed the postero-mesial area of specialised cortex. It is characterised by a deep granular layer, and by the practical absence of a supra-granular layer in the mole and shrew, although a thin representative of this layer occurs in the hedgehog. Owing to minor differences of structure this region is thought to be divisible into three parts, and it was suggested that the upper division is concerned with the cortical representation of the fifth sensory nerves, and the lower with the optic nerves.

In the best developed areas of the cortex considerable differences exist in total depth in the three animals, but these are believed to be dependent chiefly upon mechanical factors and to bear no relation to the intelligence of the animal. The *relative* depths of the separate cortical layers are approximately the same in all. Micrometric measurements of the layers are given from areas 1 and 2, etc., in the mole, and these are compared with Bolton's measurements of the developing human cortex and that of the normal human adult. [The same order of development of the cortical layers was found by the writer to occur in several fœtal and new-born lower mammals.] The chief facts shown by an analysis of these measurements are: 1. The granular layer is of approximately the same depth in the mole (areas 1 and 2) as in the normal human adult prefrontal cortex. 2. The supra-granular (pyramidal) layer; (*a*) in the sixth month human fœtus is nearly 2½ times the depth of that of the mole; (*b*) in the full time child it is six times the depth; (*c*) in the human adult nine times the depth. 3. The infra-granular cortex (layers 4 and 5), in great contrast to the supra-granular (pyramidal), is in the sixth month fœtus and full time child little over the depth found in the mole. In the human adult it is only slightly over half as deep again as in the mole (1·56 to 1). The increase in depth of the human cortex as compared with that of the mole is therefore *very* largely due to increase in the supra-granular (pyramidal) layer.

From the developmental aspect, onto- and phylo-genetic, it is

concluded that the mammalian cortex is built up on an infra-granular basis, *i.e.* the infra-granular portion is the earliest to appear, quickly reaches maturity, and in the adult presents remarkably little difference in absolute depth in one of the lowest mammals and in the highest. The granular layer is the next addition to the cortex, whilst the last to appear is the supra-granular (pyramidal) which is the slowest of all the laminæ to reach maturity, is scarcely existent in certain regions in the lowest mammals, and even at its best in the latter attains but a slight absolute depth as compared with its depth in almost every region of the human neopallium. The lowest grade of neopallial representation thus appears to be little more than a repetition of the hippocampal type—granular and infra-granular cortex. By the accrescence of a supra-granular layer of varying degrees of depth and complexity higher grades of representation are reached.

The conclusions concerning the functional significance of the cortical layers (which are supported by an examination of the neopallium of mammals belonging to other orders), form a complement to those announced by Bolton, as the result of his studies of the development of the human cortical layers, and of the depth of these in the adult normal individual, as well as in various degrees of amentia and dementia. These conclusions as regards the supra- and infra-granular portions of cortex may be very briefly stated as follows. The infra-granular cortex (layers 4 and 5, omitting the constituent cells which possess motor or analogous functions), is concerned with the associative processes necessary for the performance of the instinctive, or more or less stereotyped, animal activities. The supra-granular (pyramidal) layer subserves the higher associations, the capacity for which is shown by the degree of educability of the animal. The infra-granular layers thus constitute the earlier evolved and more fundamental associational system of the cerebral cortex, the supra-granular a higher and accessory system of later origin, and of poor development in the lowest mammals.

Note.—Some further work upon various mammals since the paper was written has led the writer to the conclusion that his first interpretation of the postero-mesial specialised area is incorrect. This area appears to be homologous with a similar peculiar cortex external to the fissura hippocampi in other mammals. The visual area of these Insectivores, if definable, must therefore be sought for elsewhere than in this region, possibly in the so-called postero-mesial undifferentiated area, although this presents in its cell lamination few features characteristic of projection cortex, perhaps owing to sub-evolution in correlation with the comparatively minute optic nerves possessed by these animals. AUTHOR'S ABSTRACT.

PHYSIOLOGY.

ON THE FUNCTIONS OF THE CEREBRUM. THE FRONTAL LOBES.

(267) By SHEPHERD IVORY FRANZ, Ph.D., *Arch. of Psychology*, March 1907.

THIS article is intended to be one of a series dealing with the functions of different parts of the cerebrum, more particularly with the so-called association areas.

After a short historical introduction, and a discussion of the various functions which have been assigned to the frontal lobes—movement, inhibition, attention, and association—the author proceeds to describe his own experiments on cats and monkeys. These consisted for the most part of observations upon the effect of experimental lesions of the frontal lobes on habits and associations which the animals had previously formed. For example, the animal learned how to escape from a box by pressing a button or pulling a string, or how to procure food by loosening a catch and opening a box.

Amongst the conclusions arrived at are the following:—

1. In monkeys and cats the frontal lobes are normally employed in the formation of simple associations.
2. When the frontal lobes are destroyed, recently formed habits are lost.
3. The loss of the associations is not brought about by lesions of other parts of the brain.
4. (Therefore) the loss of the associations is not due to shock.
5. Unilateral lesions of the frontal areas are not followed by loss, but may be followed by slowing of the motor impulses.
6. Habits lost as the result of such lesions may be re-learned.
7. Long-standing habits are retained.
8. The emotional condition of the animals seems to be unaltered.

The frontal lobes thus appear to be concerned in normal and daily associational processes, and through them we are able to form habits, and, in general, to learn.

W. B. DRUMMOND.

PATHOLOGY.

DIPHTHEROID ORGANISMS IN THE THROATS OF THE INSANE.

(268) J. W. H. EYRE and J. FROUDE FLASHMAN, *Archives of Neurology*, Vol. iii., 1907.

THE authors record some investigations planned with a view to testing the validity of the opinions advanced by Dr Ford Robertson

as to the causal association he had observed between diphtheroid bacilli (possibly B. diphtheriæ itself) and general paralysis of the insane. The technique adopted is carefully described and the material selected for investigation detailed. The authors recognise, for the purpose of classification, three types of organisms as comprising the diphtheria group of bacilli, viz. B. diphtheriæ, B. hoffmanni, B. xerosis and other unnamed species, and direct attention to the limitations, as a diagnostic test, of Neisser's method of staining. Mention is also made of the value, as an aid to differentiation, of the morphological characters which members of each of these types assume when grown upon alkaline potato.

The results of the examination of swabbings from the buccal and naso-pharyngeal mucosæ of sixty general paralytics are then detailed, analysed, and contrasted with similar material from seventy-eight cases of other forms of insanity. Stated briefly, 16·6 per cent. of the general paralytics yielded diphtheroid organisms, as compared with 17·9 per cent. of the other forms of insanity. The 16·6 per cent. of the general paralytics was made up of 5 per cent. true B. diphtheriæ, 8·3 per cent. B. hoffmanni, and 3·3 per cent. B. xerosis and other species. The 17·9 per cent. of the other forms of insanity is composed of 5·1 per cent. B. diphtheriæ, 11·5 per cent. B. hoffmanni, and 1·2 per cent. other diphtheroids, and the inference is drawn that the organism which is supposed to stand in causal relationship to general paralysis of the insane is no more readily isolated from cases of this disease than from those of other forms of insanity. An interesting point in connection with these figures, but one which the authors neglect to comment upon, is that the percentage incidence of all diphtheroid organisms in the throats of thirty-nine cases of insanity *other* than general paralysis drawn from the old asylum of Colney Hatch is nearly twice as high as it is in the thirty-nine similar cases drawn from the new building of Claybury (23·0 per cent. as compared with 12·8 per cent.). The diphtheroid bacilli isolated in the course of the work were carefully studied in cultures, and their virulence tested by inoculation upon guinea-pigs, with the result that two only were proved to be pathogenic for this animal.

In addition to the examination of the throats of cases during life, a study was made of post-mortem material from ten cases of general paralysis and twenty-six cases of other forms of insanity, special attention being paid to material from the alimentary canal and the respiratory tract. Four of the cases of general paralysis yielded B. hoffmanni post-mortem from the respiratory tract—one showing signs of a terminal infection. Of the remaining six cases, two which during life had yielded diphtheroid bacilli from the tonsils or pharynx gave absolutely negative results post-mortem. From the result of their investigations the authors fail

to find any evidence of a causal association between diphtheroid bacilli and general paralysis. AUTHOR'S ABSTRACT.

ON THE DETECTION OF SYPHILITIC ANTIBODIES IN THE
(269) **CEREBRO-SPINAL FLUID OF GENERAL PARALYTICS BY MEANS OF WASSERMANN-PLAUT'S METHOD OF COMPLEMENT DEVIATION. (Über den Nachweis syphilitischer Antikörper im Liquor cerebrospinalis von Paralytikern nach dem Wassermann-Plaut'schen Verfahren der Komplementablenkung.)** J. MORGENROTH and G. STERTZ, *Virchows Archiv*, Bd. 188, 1907, p. 166.

THE cerebro-spinal fluid of 25 patients was examined for syphilitic antibodies by means of the Wassermann-Plaut method, which is based upon the well-known procedure introduced by Bordet and Gengou for detecting an immune body in a serum. The elements employed by the authors were: (1) an extract of liver or spleen from a syphilitic fœtus; (2) cerebro-spinal fluid; and (3) guinea-pig's complement. The mixtures containing those elements were left for three-quarters of an hour, and then sheep's blood and an appropriate hæmolytic immune body or amboceptor were added. If no hæmolysis occurred, the test showed that the cerebro-spinal fluid contained syphilitic antibodies. By such means those antibodies were detected in the cerebro-spinal fluid of all the eight cases of general paralysis examined, and in that of one case of secondary syphilis presenting optic neuritis, whereas in all the other cases, including two examples of cerebral syphilis, negative results were obtained. W. T. RITCHIE.

POST-MORTEM CHANGES IN THE ENDOCELLULAR RE-
(270) **TICULUM, AND OF THE LONG FIBRILS IN THE CELLS OF THE SPINAL CORD. (Le alterazioni cadaveriche del reticolo fibrillare endocellulare e delle fibrille lunghe nelle cellule del midollo spinale.)** MATTEI, *Riv. Speriment. di Fren.*, F. 1, 1907, p. 34.

THE author has studied the degree of resistance of the fibrillar reticulum of the nerve cells (anterior cornua of cord) to putrefactive changes. The cords of oxen and rabbits were taken immediately after death, allowed to putrify, and examined at intervals varying from one to ten days. The changes shown by Donaggio's method (iii.) were as follows: Imperfect differentiation of the nucleus, vacuolation of the nucleolus, breaking up of the endocellular

reticulum, thickening of the long fibrils at the cell periphery, rarefaction of the perinuclear chromatic substance, disappearance of fibrils and protoplasmic processes, and formation of granular detritus. It is to be noted that, according to Donaggio's method, the endocellular reticulum and long fibrils remain normal almost up to the second day after death. DAVID ORR

PRIMARY LESIONS OF THE SPINAL NERVE FIBRES. (Lesioni
(271) **primarie delle fibre nervose spinali.)** RIVA, *Riv. Speriment. di Fren.*, F. 1, 1907, p. 258.

FOR this research adult animals were used. Some were starved to death, others exposed to acute intoxication from picrotoxin and absinthe; one was killed by exposure to cold. The cords were examined by Donaggio's method for changes in the cells and fibres, and no relationship was found between the lesions in these.

As to the lesions of the white matter in the cord, the dorsal region was always most affected and showed greater variety of localisation, the cervical region showed less affection and in a more uniform manner, while the lumbar region was generally intact. The columns which were less resistant to the toxic action were the crossed pyramidal and the posterior; the former more especially in the cervical region, the latter in the dorsal. The lesions were always bilateral, and, even when diffuse, symmetrical. In the posterior columns the degenerated fibres occupied zones of varying form, according to the level at which they were situated, but were especially noted in Flechsig's postero-median root zone. The central portion of Goll's tract was usually left intact, and sometimes the central oval of Flechsig.

It would seem as if the toxin produces, by its action on the fibres, lesions which correspond topographically, especially in the posterior columns, to the fœtal distribution of the spinal fasciculi.

DAVID ORR.

CLINICAL NEUROLOGY.

MYOPATHY FOLLOWING TYPHOID FEVER. (La myopathie
(272) **consécutive à la fièvre typhoïde.)** GUILLAIN, *Sem. méd.*, June 12, 1907, p. 276.

A COACHMAN, aged 45, free from hereditary or personal taint, contracted a severe attack of typhoid fever, in which intestinal hæmorrhage and a relapse took place. On leaving hospital in March 1901 he had some weakness of the lower limbs, but could

walk without assistance. During the next five years this weakness gradually increased, and the upper limbs became involved. The muscles also became progressively atrophied. There was no pain nor any disturbance of sensation, and the sphincters were not affected. He pursued his calling till May 1906. In January 1907 most of the movements of the limbs and trunk were performed with great difficulty, or were quite impossible, and the muscular atrophy was very marked. Lordosis was pronounced, and the gait was waddling. In raising himself from the floor he adopted the characteristic method of climbing along his legs and thighs. The knee and ankle jerks were lost, but the cutaneous reflexes were normal. There were no sensory nor mental changes. The muscles did not present the reaction of degeneration, but a mere diminution of faradic and galvanic excitability.

The existence of myopathy following typhoid fever is of interest, not only because such cases are very rare, but because their existence proves that side by side with the hereditary and familial myopathies there are also acquired myopathies. The progressive myopathy following typhoid may fitly be compared to the chronic myocarditis which sometimes develops in the disease, both being due to the same toxine.

J. D. ROLLESTON.

NEURO-MYOSITIS FOLLOWING VARICELLA. (**Un cas de névro-myosite post-varicelleuse.**) (273) CAMY et SÉZARY, *Rev. Neurol.*, April 30, p. 393 (Soc. de neurol.).

A GIRL, aged 11, belonging to a neuropathic family, developed varicella in 1901. Three days after the onset the legs became completely flexed upon the thighs, and could not be straightened without causing very severe pain. A few weeks later extension became more easy, but shooting pains rendered walking and the upright position impossible. The legs atrophied, and the upper limbs soon became similarly affected. A prolonged course of electricity and massage was followed by only slight improvement. On admission to Déjerine's service at the Salpêtrière, in June 1906, the child was much emaciated, and showed a general atrophy of all the muscles. Walking was difficult, and the power in the upper limbs was very weak. Complete extension of the legs was rendered impossible by retraction of the flexors of the thigh. These muscles were rigid, presented a fibrous consistency, and were tender on pressure. The joints were healthy. The knee and ankle jerks were almost abolished on both sides. In the upper limbs there was an atrophy of the different muscle groups which were similarly tender on pressure. The biceps on each side showed a very marked retraction and a fibrous consistency.

The superficial nerves were exceedingly tender on pressure. Tactile sensation was preserved. The face was unaffected, and there were no ocular troubles. In the course of eight months considerable improvement took place. Under the influence of massage and mobilisation the retraction of the lower limbs disappeared, and walking became almost normal. The improvement in the upper limbs was less marked. Electrical examination showed only a diminution of the contractions without modification of the formula.

In spite of the long duration of the affection the diagnosis of polyneuritis was confirmed by the generalisation of the motor symptoms, the tenderness of the nerves, the disturbance of the reflexes, and the slow but progressive improvement.

The peculiar consistency of the muscles, their electrical reactions, and above all the pronounced fibrous retraction appeared to be the result of a cicatrisation of a subacute interstitial myositis. The intense hyperæsthesia of the muscles confirmed this view.

J. D. ROLLESTON

A CASE OF DISSEMINATED SCLEROSIS. F. RAYMOND, (274) *L'Encéphale*, Vol. ii., No. 3, p. 225.

A MAN of 26. The disease started with weakness in the lower limbs from no apparent cause, and progressed steadily with one short remission, causing death in about two years. He had intention tremors, his speech was affected, there were ocular disturbances, and his sphincters were early and markedly affected. There was a striking lymphocytosis of the cerebro-spinal fluid and a marked œdema of the lower limbs, apparently trophic in nature.

Microscopically the case was one of *sclérose en plaques* with some unusual features. Around the sclerosed patches there were more or less diffuse lesions with nerve cell degeneration, axis-cylinder destruction, but no sclerosis. There were also areas of recent change where the myelin still stained by Marchi's method; in parts of these the axis-cylinders were also much altered, but there was little or no neuroglial proliferation.

Vascular phenomena were evident if not just striking, thickening of vessel walls, obliteration of lumen in a few, new vessel formation in the areas of recent sclerosis, and greater neuroglial proliferation around the vessels than elsewhere.

It could not be affirmed whether the sclerosed patches and the surrounding diffuse lesions were due to the same cause (possibly a toxic one, from the vascular changes), or whether the case should be looked upon as one of disseminated sclerosis with added lesions of secondary myelitis.

J. H. HARVEY PIRIE.

NERVE ROOT AND GANGLION COMPRESSION IN CASES OF
(275) **BRAIN TUMOUR.** P. LEJONNE, *L'Encéphale*, Vol. ii., No. 3, p. 232.

BASING his observations on 15 cases of brain tumour, the author describes a syndrome which may be called indifferently *la compression radiculo-ganglionnaire* or *radiculo-medullaire*. Clinically the symptoms may be almost latent, and in any case are only to be seen before they are masked by the symptoms of direct cerebral pressure. Slight cases complain of some pain in the back of the neck, there is usually some paræsthesia in the arms or legs, and slight diminution of the tendon reflexes, especially in the lower limbs.

When more marked, the knee-jerks may be entirely lost, there is often slight ataxia, particularly in the hands; subjective sensory disturbances are more pronounced, there are vaguely defined areas of hypoæsthesia, and there is usually diminished vibration sensibility. The pressure of the cerebro-spinal fluid is raised to about 25 or even 40 cm. of H_2O, higher than it is in any other chronic nervous disease.

The dural canal around the spinal nerve roots can be seen to be dilated. The primary lesion is found to be a separation of the posterior root fibres, and even of the proximal portions of the root ganglia, by intrusion of cerebro-spinal fluid into the arachnoid cul-de-sacs between the fascicles. There is some engorgement of veins, but no sign of any inflammatory reaction.

The fibres of the posterior roots show granular disintegration of the myeline sheaths, and in advanced cases, swollen, pale, and vesicular, but always persistent axis-cylinders. Within the cord the lesions are similar, though less pronounced, and are limited to the posterior root zone.

The author differs from Nageotte in ascribing these changes and the resultant symptoms to purely mechanical causes, viz. the increased pressure of the cerebro-spinal fluid. He has attempted experimental confirmation, but so far has not succeeded in maintaining a permanently high pressure within the cerebro-spinal fluid.

J. H. HARVEY PIRIE.

CEREBRAL ABSCESS FOLLOWING MEASLES. (Abcès cérébral
(276) **consécutif à la rougeole.)** COLLET, *Lyon médical*, June 2, 1907, p. 1025.

A GIRL, aged 9, shortly after an ordinary attack of measles, developed cerebral symptoms, manifested by headache, somnolence, and dilatation of the pupils. Meningeal symptoms were absent.

The temperature first showed irregular oscillations, and then hypothermia. Coma and death ensued, and the autopsy showed an enormous cerebral abscess in the left frontal lobe without any meningitis. Cerebral abscesses in measles are almost always secondary to lesions of the middle ear. In this case, however, there was no trace of otitis media. The brain was probably infected either through the blood stream or through the nasal lymphatics, an abundance of muco-pus being found in the ethmoidal cells. The hypothesis of the abscess being tuberculous is set aside by the absence of tubercle in the lungs and of enlargement of the bronchial glands. J. D. ROLLESTON.

INFANTILE CEREBRAL PARAPLEGIA AND INFANTILE (277) SPINAL PARALYSIS IN THE SAME PATIENT. (Coincidence chez un même malade de la paraplégie cérébrale infantile et de la paralysie spinale infantile. Autopsie.) ITALO ROSSI, *Nouvelle Iconographie de la Salpêtrière*, March 1907, pp. 122-145.

THIS valuable paper contains the account of a case of the above nature that was under observation for ten years. The patient, aged 30, had suffered from paraplegia that came on suddenly, with convulsions, at the age of six months. The right lower extremity was in a state of spastic paralysis, the left of flaccid. On the left side, which was very atrophied, Babinski's sign was the only indication of any pyramidal affection. At the autopsy there was found in the brain on both sides a focus of softening, affecting the inner and upper part of the first frontal convolution, and the front half of its orbital portion, the inner surface of the paracentral lobule, the first limbic convolution, and the front three quarters of the corpus callosum. In the cord there was a typical focus of old poliomyelitis in the left anterior horn, extending from the first lumbar segment to the second sacral, with marked atrophy of the corresponding nerve roots. The case is described in great detail, and the account is profusely illustrated by photographs.

Six similar cases have previously been described, though only one of these was followed by autopsy. The obvious bearing of the observation on Vizioli's hypothesis of the identity of poliomyelitis and certain types of encephalitis is fully discussed, as also the other evidences that support this view. An extensive bibliography is given. ERNEST JONES.

CHRONIC POLIOENCEPHALITIS: OPHTHALMOPLEGIA AND
(278) **BILATERAL PARALYSIS OF THE MOTOR DIVISION OF THE FIFTH NERVE. PROBABLE TABES. (Polioencéphalite chronique: Ophtalmoplégie et paralysie bilatérale de la branche motrice du trijumeau. Tabes probable.)** HENRI LAING, *Nouvelle Iconographie de la Salpêtrière*, March 1907, p. 117.

THE patient, who was shewn before the Société de Neurologie, was a woman of 49. The symptoms in question had all come on suddenly and simultaneously a year before. Both third nerves were totally paralysed, the pupils were small, and Argyll Robertson's sign was present on both sides. There was bilateral paralysis of the motor division of the trigeminal, and its electric excitability was lost. The mouth could be closed by narrowing the orbicular orifice. Mastication was carried out only by to and fro movements of the jaw, performed by the hyoid muscles. The left knee-jerk was abolished, and the right obtained only by Jendrassik's method. There exist in the literature accounts of three similar cases. ERNEST JONES.

IS EPILEPSY A DISEASE OF METABOLISM? A REVIEW OF
(279) **THE LITERATURE.** J. F. MUNSON, *Journ. Nerv. and Ment. Dis.*, May 1907.

THE writer doubts the presence of an actual gross organic lesion as the cause of epilepsy, and believes that the epileptogenous change affects the chemical structure of the cell. Bio-chemical methods of research are those most likely to throw light on the etiology of epilepsy. The work already done on such lines concerns the urine (especially with regard to phosphorus metabolism and urine toxicity), the sweat, the gastric juice, the blood, and the cerebro-spinal fluid (the discovery of the presence of cholin in cases of epilepsy may prove to be of importance). As yet, the findings by different observers have been very contradictory, but the fact that some variation from the normal is almost always reported, makes one hopeful that the problem may ultimately be solved by work of this nature. HENRY J. DUNBAR.

SOME SUBSTANCES WHICH ENTER INTO THE GENESIS OF
(280) **THE EPILEPTIC SEIZURE.** DONATH, *Ann. Méd.-Psych.*, May-June 1907, p. 353.

GRANTING the general predisposing causes of idiopathic epilepsy, one has still to ascertain the actual existing factors in the individual attacks, and these, it would appear, are chemical irritants

produced during the metabolic changes of the organism itself. In order to determine which substances are capable of producing convulsions, the author has performed a series of experiments on dogs, the details of which he gives in the present article. The administration of the substances was carried out by intravenous injection. Uric acid in the form of neutral sodium urate caused no convulsions. Moreover, excess of uric acid in the blood occurs in gout and in leukæmia, and yet in neither of these diseases do convulsive attacks take place. The diminished excretion of uric acid before and the proportionately increased excretion after an epileptic fit are due, not to a disappearance of uric acid by reason of the formation of ammonium carbonate, but simply to a retention of uric acid in the blood. Further experiments show that lactic, acetic, oxybutyric, and mineral acids do not act as convulsants. As far as the experiments have gone, the only substances found to be capable of producing convulsive seizures—and these do so very definitely—are ammonia and the ammoniacal organic bases, such as trimethylamin, cholin, creatinin, and guanidin. The vascular changes found in the brains of epileptics are considered by the author to be secondary to the enormous increase of the cerebral blood pressure during the epileptic attacks. HENRY J. DUNBAR.

A CASE OF EPILEPSY ASSOCIATED WITH ACROMEGALY.
(281) W. T. SHANAHAN, *Journ. Nerv. and Ment. Dis.*, May 1907.

THE patient was a married woman, aged 31 years, who had exhibited symptoms of acromegaly for nine years, during the last five of which numerous epileptic seizures, apparently grand mal in type, had occurred. The frequency of the attacks had varied from one in two days to one in three weeks. The left side had been noted to be first involved in some fits. Examination revealed typical symptoms of marked acromegaly; and since coming under observation the patient had had numerous petit mal seizures which began in the left hand as if she were trying to approximate the finger tips. Grand mal attacks had also occurred, but had not been satisfactorily observed. The writer reviews several other published cases showing the same combination of symptoms, but considers that in some of them the epilepsy was merely of the symptomatic Jacksonian type accompanying brain tumour. Apart from the presence of a pituitary tumour which would cause epileptoid symptoms by direct pressure on the cortical substance or by general increase of intracranial tension, epilepsy in acromegaly might be produced by a perverted pituitary secretion acting on unstable tissues, or by such a secretion or lack of secretion allowing the action of other toxins. HENRY J. DUNBAR.

CLINICAL CONTRIBUTION TO THE STUDY OF THE EPI-
(282) **LEPTIC EQUIVALENT (TRANSITORY MANIA).** MONDIO, *L'Encéphale*, April 1907, p. 419.

THE author was asked to determine the mental condition and criminal responsibility of a soldier, aged 26 years, accused of insubordination with violence and insults to his superiors. Following an attack of syphilis, he underwent a period of mental depression, culminating in a determined attempt at suicide, which he subsequently declared he had no recollection of. Shortly afterwards the offences in question were committed, and on being questioned after his arrest he gave a complete denial, and stated that the charge had been maliciously invented. On examination it was found that the cranium was small, and that he suffered from repeated headaches and disorders of sensibility. He was easily tired, and exhibited symptoms of hypochondriasis and melancholia. Separately considered, the first outburst—suicidal attempt—might be termed an epileptic equivalent, and the second—the insubordination—an attack of transitory mania. The author, however, argues that these conditions are identical. He considered that the man was entirely irresponsible criminally, and his conclusions being accepted by the Tribunal, a sentence of acquittal was pronounced.

HENRY J. DUNBAR.

THE PSYCHICAL MANIFESTATIONS OF MIGRAINE. (I fenomeni
(283) **psichici nell' emicrania.)** V. FORLI, *Rivist. Sper. di Freniat.*, Vol. xxxiii., Fasc. 1, pp. 220-241.

FORLI has examined 185 cases of migraine in the Rome Psychiatry Clinic during the past few years, and on the basis of this study deals with some of the problems of the condition. His main conclusions are these :—

1. In connection with migraine attacks, psychical disturbances are not rare ; they are ordinarily slight. They may occur one or even two days before the attack ; they may immediately precede it, constituting an aura ; or they may occur only as the pain is subsiding. More frequently they begin at the acme of the attack and then gradually diminish. In spite of the last fact one should not conclude, as Griesinger did, that the disturbances are dependent on the pain, but rather that both are separate symptoms of a common cause. When this is in great activity both symptoms will be marked.

2. The psychical disturbances relate to the whole field of mental activity, but certain psycho-sensorial phenomena predominate, especially in connection with sight and hearing. A

whole series can be followed from the simplest hallucinations to the most complex.

3. The latter phenomena were met with in cases in which hysteria and epilepsy could be completely excluded; they were exclusively in relation with the migraine. A special feature of the hallucinations is that they exercise no influence on the conduct of the patients.

4. Migraine is in most cases a manifestation of an independent affection. Symptomatic migraine may occur in tabes, cerebellar lesions, epilepsy, etc. True migraine resembles epilepsy only inasmuch as both affections develop on a neuropathic basis; they have a separate genesis and a different evolution.

ERNEST JONES.

ON THE RENAL SECRETIONS IN MIGRAINE. (Sul Ricambio
(284) **urinario nelle Emicranie.)** M. A. BIOGLIO, *Rivist. Sper. di Freniat.*, Vol. xxxiii., Fasc. 1, p. 49.

AFTER mentioning the researches that various workers have carried out on the urine of epileptics, the author gives in detail the results of some observations on four cases of migraine. The conclusions he arrives at are stated at length, but may be summarised as follows:—

A. During the interval periods: the nitrogenous excretives are slightly diminished, the chlorides, sulphates, and earthy phosphates are increased, though the total phosphatic excretion remains normal.

B. During the attacks: the nitrogenous excretion is increased, all the other constituents remain normal or vary irregularly.

ERNEST JONES.

SUDDEN NERVOUS ACCIDENTS IN AUTOMOBILISM. (Disturbi
(285) **nervosi improvvisi e infortuni d'Automobilismo.)** PIETRO PETRAZZANI, *Rivist. Sper. di Freniat.*, Vol. xxxiii., Fasc. 1, pp. 150-172.

THIS paper was written apropos of some recent accidents, which are here described, to call attention to the increasing responsibility resting on the drivers of motor-cars. The author lays special stress on various visual anomalies, particularly astigmatism and partial spasm of accommodation, in relation to such accidents. Accommodator spasm, which he considers is a quasi-professional defect, may supervene at a critical moment with disastrous results. He urges that the legislature should enforce careful medical examination of all drivers.

ERNEST JONES.

PAINFUL ADIPOSIS IN AN EPILEPTIC AND BLIND IMBECILE. (Adipose douloureuse chez une imbécile épileptique et aveugle.) (286) A. PRUNIER, *Nouv. Iconogr. de la Salpêtrière*, March 1907, p. 168.

THE patient was a woman, aged 54, who had been in the asylum since the age of ten, on account of epilepsy and imbecility; the latter was very marked. Pronounced adiposis supervened at the age of 49. The masses were widely generalised and affected even the face, a rare situation; in the limbs sharp lines separated the enlarged parts from normal hands and feet. The masses were very tender to pressure and to movement. ERNEST JONES.

CHRONIC TRAUMATIC TROPHŒDEMAS. (Des trophœdèmes chronique d'origine traumatique.) (287) G. ETIENNE, *Nouvelle Iconographie de la Salpêtrière*, March 1907, p. 146.

ETIENNE discusses the condition called trophœdema by Meige, and defined as a chronic, harmless, painless, white, hard œdema of segmental distribution. The cases can be clearly divided into two classes, the hereditary and the acquired. Etienne now adds two cases of traumatic trophœdema to the few that have previously been recorded. In both there was present a sensory ascending neuritis due to prolonged trauma.

Etienne's view as to the pathogenesis of the œdema is that the sympathetic nervous system is primarily at fault in the hereditary cases, whilst in the traumatic cases it is affected secondarily to the trouble in the lowest centripetal neurone. He thinks that irritative vaso-dilatation is more important than paralytic vaso-constriction in these cases. ERNEST JONES.

CASE OF CHRONIC TROPHŒDEMA. (Sur un cas de Trophœdème chronique.) (288) G. PARHON and S. FLORIAN, *Nouv. Iconogr. de la Salpêtrière*, March 1907, p. 159.

THE authors record the case of a girl of 19, whose symptoms date back six years. The left thigh, leg and foot—though not the toes—were enormously enlarged. The limb was not tender, and and did not pit on pressure; its colour was natural. The onset was rather sudden with pain, fever, and a vesicular eruption. The enlargement began on the thigh. There was no similar case found in the family.

A good general account of trophœdema and a short review of the published cases concludes the article. ERNEST JONES.

HISTOLOGICAL EXAMINATION OF THE INTEGUMENTS AND
(289) **NERVOUS TRUNKS IN A CASE OF CONGENITAL TROPHŒDEMA. (Examen histologique des téguments et des troncs nerveux dans un cas de Tropœdème congénital.)** E. LONG, *Nouv. Iconogr. de la Salpêtrière*, March 1907, p. 155.

A FULL clinical description of the case in question has been published by Rapin in the same journal in 1905; the case belongs to Meige's group of hereditary trophœdemas. The patient, a woman of 30, had suffered with a hard œdema affecting both upper limbs of the right cheek and ear. The right arm was amputated four years ago on account of a cutaneous cancer, and Long now relates the results of microscopic examination of it. The hard "œdema" was chiefly due to a fatty layer, one centimetre thick, beneath the dermis. There was also a considerable fibrosis in the dermis and deeper structures. The proper constituents of the nerves, blood-vessels, and muscles were quite normal.

ERNEST JONES.

ISOLATED PARALYSIS OF THE SERRATUS MAGNUS. CLAUDE
(290) and DESCOMPS, *L'Encéphale*, Jan. 1907, p. 28.

THE patient, a tailor aged 35 years, well developed and in excellent health, had suffered for about two and a half months from severe pain in the right shoulder and arm, with diminution of muscular power in the right arm and "winging" of the right scapula. On examination he was found to present the typical signs of serratus magnus paralysis with regard to the electrical reactions, the deformities of the shoulder and scapula in the various positions of the arm, and the deformity of the thorax. The writer emphasises specially two signs which have been neglected in most of the previously published cases. Firstly, the muscular power of the whole of the right arm was found to be diminished. This is not the result of actual muscular weakness of the arm itself, but is due to the want of fixation of the scapula, as was proved in this case by binding the scapula firmly to the chest wall by means of a bandage, when it was discovered that the power of the arm was normal. Secondly, it was noted that scoliosis was present, especially when the arms were raised vertically above the head. Two curves were demonstrable, the one cervico-dorsal with the convexity to the left, the other dorso-lumbar with the convexity to the right. In addition, there was present a cervico-dorsal kyphosis with a lumbar lordosis. These deformities are illustrated by photographs. The author discusses in detail the mechanism of the spinal curvatures. One symptom complained of by the patient has some

bearing on the question of the etiology of the paralysis—namely, cracklings in the region of the shoulder-blade on movement of the arm. Such cracklings could be produced by an inflammation of the subscapular bursa, which lies on the posterior thoracic wall in the angle formed by the convergence of the subscapularis and serratus magnus muscles. The nerve of Bell lies closely upon this bursa, and any inflammatory condition of the latter would readily affect the former and set up a neuritis. HENRY J. DUNBAR.

ON THE PUPILLARY REACTION TO PAIN AS A DIAGNOSTIC
(291) **POINT BETWEEN ORGANIC AND PSYCHOGENIC TENDERNESS. (Ueber die Schmerzreaktion der Pupillen als ein differential-diagnostisches Zeichen zwischen organischer und psychogener Druckschmerzhaftigkeit.)** M. LOWY (of Marienbad), *Neurol. Centralbl.*, Oct. 16, 1906.

PUPILS, which are contracted under bright light, dilate on painful pressure over any affected region if there is an organic affection, but do not dilate if the pain or pressure is psychogenic.

C. MACFIE CAMPBELL.

ECTOPIA PUPILLÆ IN CERTAIN MESENCEPHALIC LESIONS.
(292) S. A. K. WILSON, *Brain*, 1906, p. 524.

WILSON has found ectopia pupillæ to supervene during the course of not a few cases of disease in the mid-brain. Details of three such cases (with autopsy in two) are given, in which he could exclude any congenital anomaly or the operation of any cause of an extraneous nature, such as old inflammation. The deviation of the pupil in each case was upwards and inwards. The salient features of the cases are these.

In the first, double symmetrical corectopia supervened in the course of a pontine tumour at least four weeks before death and continued to the end. Clinically there was considerable impairment of ocular movement, and pathologically there was evidence of involvement of the oculo-motor nerves.

In the second, a patient with indications of intracranial growth had peculiar congestive attacks in which he became unconscious for hours, during which ectopia pupillæ was noted on both sides. It disappeared during one of these attacks, but reappeared during the final one, which was fatal. Ophthalmoplegia was present, and a tumour was subsequently discovered occupying the whole of the third ventricle and pressing down and back on the upper part of the pons.

In the third, which could scarcely be other than disseminated sclerosis, ectopia pupillæ appeared in hospital, when there was definite defect of ocular movement, and about thirty-six hours before the development of an incomplete oculo-motor palsy. It continued for a month or more, and improved, although it did not entirely disappear, with improvement in the other ocular symptoms.

Wilson refers briefly to the physiology of pupillary movement, and gives reasons for believing that the ectopia pupillæ in his cases was due, not to changes in the ciliary ganglia, but to disturbances in the upper part of the pons—a feature of all the cases. He suggests that, since there was no gross interference with pupillary reactions, the cause was mainly a vasomotor one, due to disturbance in the function of vaso-dilator and vaso-constrictor fibres which are closely connected in their central origin.

A. W. MACKINTOSH.

ON THE MOVEMENTS OF THE TONGUE IN HEMIPLEGIA

(293) **AND FROM CORTICAL STIMULATION—AN APPARENT PARADOX; WITH A NOTE ON THE MOVEMENTS OF THE TONGUE AFTER PARALYSIS OF ONE HYPOGLOSSAL NERVE.** CHARLES E. BEEVOR, *Brain*, 1906, p. 487.

"IN hemiplegia the tongue, when affected, is protruded towards the paralysed side, or, in other words, towards the side opposite to the lesion in the internal capsule or the cortex. On the other hand, electrical stimulation of the cortex cerebri in the monkey produces protrusion of the tongue to the side which is opposite to the cortex stimulated. Assuming that we may compare the brain of the monkey with that of man, we apparently have the same movement of protrusion of the tongue to the opposite side produced by both paralysis and stimulation of the cortex, which sounds an absurdity."

As a matter of fact, the movement is not the same in the two cases. Horsley and Beevor have shown experimentally that the cortical "tongue area" of the monkey is divided into two parts. In the upper part is represented the movement of protruding the tongue towards the opposite side; in the lower part, stimulation of the anterior half gives the movement of advancing the tongue straight, while in the posterior half we obtain the movement of retraction of the tongue. These two movements—protrusion of the tongue straight and protrusion to the opposite side—are quite distinct in character. When the tongue was divided into two halves down to the hyoid bone, stimulation of the area (in one hemisphere) for

protrusion of the tongue straight caused both halves of the tongue to be simultaneously and equally protruded straight—this movement is truly "bilaterally represented"; when, however, the upper part of the tongue area in the left hemisphere was stimulated, the left half of the tongue was advanced and the right half retracted—the resultant of these two movements on an entire tongue being, naturally, protrusion to the right side.

The experiments showed that, in every movement of the tongue produced by cortical excitation, both halves of the tongue always take part. It is difficult to explain why, in hemiplegia, the movement of protruding the tongue straight is ever interfered with. Beevor suggests that both hemispheres take part in this movement, and that, although both halves of the tongue are represented in each hemisphere, yet in voluntary efforts, when one hemisphere is unable to act (*e.g.* in hemiplegia), the other acts more strongly on the opposite half of the tongue and carries it over towards the paralysed side.

In right hemiplegia there is more difficulty in putting the tongue into the right cheek than into the left; this is only natural, if the deliberate movement of protruding the tongue to the right is produced by the left cortex.

In a case of paralysis of the left hypoglossal nerve, an attempt to protrude the tongue straight brought the tip a little to the left of the middle line to about the level of the left canine tooth; owing to paralysis of retraction of the left half of the tongue, there was no power of putting the tongue into the left cheek; an attempt to protrude the tongue to the right caused the tip to be twisted round (by retraction of the right half) so as to touch the right lower jaw as far back as the second molar, but the tongue could not be protruded beyond the teeth, owing to paralysis of advancement of the left half of the tongue.

A. W. MACKINTOSH.

PSYCHIATRY.

ON MENTAL DISEASES IN THE RUSSIAN ARMY DURING
(294) **THE RUSSIAN-JAPANESE WAR. (Ueber Geisteskrankheiten im russischen Heer während des russisch-japanischen Krieges.)** M. SCHAIKEWICZ.

CRITICAL REMARKS ON THE COMMUNICATION OF SCHAI-
(295) **KEWICZ.** W. STIEDA, *Centralbl. f. Nerv. u. Psych.*, Nov. 15, 1906.

IN the first communication the author describes, under the title of Amentia Depressivo-stuporosa, a form of mental disorder very frequently observed in the soldiers under his care during the war,

but which he had often seen in soldiers in time of peace. He notes the onset of psychoses in close relation to the actual shock of battle, and refers to severe hysterical deaf-mutism resulting from the shock of bursting shells.

Stieda denies the specific character of the above-mentioned amentia, and holds that the cases really belong to dementia præcox or manic-depressive insanity; war does not produce any specific form of mental disorder. In many of the cases of transitory deaf-mutism the line between a functional and an organic disorder could not be drawn. Neurasthenic and hysterical symptoms were common among the officers, even without trauma. He notes the presence in many soldiers convalescent from typhoid of a very short period during which the patients, even although not previously delirious, produced rather dilapidated ideas of grandeur, and showed a tendency to confabulation; after three or four days this simmered down and the patient had good insight into the absurdity of his previous remarks. The author calls attention to the analogy between this disorder and the polyneuritic psychosis.

C. MACFIE CAMPBELL.

Review

TREATMENT BY HYPNOTISM AND SUGGESTION. C. LLOYD TUCKEY, M.D. Fifth Edition. Baillière, Tindall & Cox, London, 1907. 10s. 6d.

A RENEWED study of Dr Lloyd Tuckey's work in its fifth edition reminds British students of hypnotism of the extent of their indebtedness to the author. To all who have sought accurate knowledge of the labours of the Nancy school he has been a most efficient guide. To most of us he was the pioneer who pointed out the way, and he still leads us as the president of the new Medical Society for the study of suggestive therapeutics.

It will be generally agreed that Dr Tuckey has given us a very admirable text-book. While he supplies abundant proof of the practical value of treatment by suggestion, we find in his pages no exaggerations, only absolutely trustworthy facts. If some of the incidents recorded appear to one who is commencing the study more strange than probable, the more experienced student will probably recognise these as classical examples, which he now regards as not only true, but somewhat commonplace; experiments which have been tested and repeated till their respectability has almost made them uninteresting. But surely in a text-book on such a subject respectability of this kind is a quality much to be

desired. Strictly truthful in detail, and modest in theory, Dr Lloyd Tuckey gives to a medical practitioner or student who wishes to know about psychotherapeutics exactly what he wants, and most probably what is best for him. He is not introduced into a realm where previous scientific training will seem out of place. This is pre-eminently a scientific study of hypnotism, and as such, an appeal to members of a scientific profession which they can no longer honestly ignore. The evidence adduced for the therapeutical value of hypnotism is overwhelming, and all possible disadvantages and limitations have been fully and judicially dealt with.

The contents are well arranged, and we find little to criticise in the way Dr Tuckey has carried out his task. We notice a few repetitions which might perhaps have been avoided in a fifth edition, but occasional repetition may be excused to one who has such good reason for valuing the reiteration of suggestions; and those who have come to regard a certain patient of Dr Milne Bramwell as almost a familiar friend, will doubtless forgive Dr Tuckey for varying estimates of the exact number of heroic attempts made to hypnotise her (*cf.* pp. 168, 229).

While thus strongly recommending Dr Tuckey's volume as a text-book, and recognising that its very limitations may increase its popularity and usefulness, we cannot but regret that, like Dr Milne Bramwell, Dr Tuckey should have been so ready to draw a boundary line which appears to us wholly artificial. I refer to his rejection of all mesmeric theories and of the more obscure phenomena of his science; a rejection which sometimes leads him into inconsistencies. An example may make our meaning clearer. Referring to the sensation of warmth produced by suggestion accompanied by the application of the hand to the epigastrium, Dr Tuckey says (p. 56): "The magnetisers attributed it to the passage of magnetic fluid from them to the patient; but, as we regard all the manifestations of hypnotism as subjective, we must of course seek another explanation. This is found by supposing that the sensation is due to action on the vaso-motor system through the solar plexus, allowing a sudden afflux of blood to the part.

This supposition is ingenious, but hardly satisfactory. The idea of a magnetic fluid is indeed shocking to the modern scientific mind—the words do sound a little mediæval; but why the application of the operator's hand over the solar plexus, and the consequent action on the vaso-motor system, should be more in harmony with the subjectivity of all the manifestations of hypnotism, is, to say the least, perplexing. Dr Tuckey would probably answer that it is the mental suggestion which is alone important. But this only leads to the further question, What is suggestion?

What happens when, as Dr Tuckey admits (having come, he tells us, to the conclusion with great unwillingness), an operator has occasionally sent a subject to sleep at a distance?

Dr Tuckey is more consistent, and presumably more happy, when he tries to explain away manifestations of clairvoyance on the part of hypnotic subjects. Here many will disagree with him; some of us being inclined to think that a certain amount of clairvoyance can be induced in a fair proportion of somnambules. Any picking and choosing between the phenomena we should like to believe, is surely to be deprecated. The late Mr Ernest Hart tried to do this. He made a few simple experiments in hypnotism, accepted these as genuine, and denounced all the more advanced work of his medical brethren as fraud. Compare Dr Tuckey's critical attitude towards the more obscure phenomena with his somewhat facile adoption of rather crude physiological statements such as this on page 144.

"All are agreed that delicate modifications in the cerebral circulation constitute the basis of the hypnotic phenomena." Dr Tuckey quotes Myers' saying that psychical processes can never be explained by physiological theories, and adds, "The physiologists will not readily admit that proposition." Perhaps not; still, dependence on physiological theory alone must have a narrowing effect in a research of this kind. Many will prefer to Dr Tuckey's own attitude that of Sir Francis Cruise in the interesting introductory chapter, where he deprecates attempts at explanation, and quotes the saying, "Nature properly interrogated tells us much of the how, when, and where; but when asked why, she is silent."

We believe that all attempts to make hypnotism a simple, well-defined branch of natural science must fail, reassuring as such attempts must sometimes be to the minds of nervous and suspicious patients. We are grateful to Dr Lloyd Tuckey for mapping out so carefully this little corner territory of psychotherapeutics; but our cultivation thereof will not be wholly satisfactory until we make bolder excursions into wider fields, which may be already ripening for harvest. GEORGE KERR.

Proceedings of Societies

AT a stated meeting of the New York Psychiatrical Society, held on March 6, 1907—Allan M'Lane Hamilton, M.D., in the Chair —Dr August Hoch read the paper of the evening, the subject being, "The Psychogenetic factors in some paranoic conditions, with suggestions for prophylaxis and treatment."

Dr Hoch pointed out that among the paranoic states there were cases, and that they probably represented a large proportion, in which the psychogenesis could be clearly traced, when the facts of the cases were really accessible. The theory of the development of paranoic states Dr Hoch summarised briefly as follows, stating that besides basing his ideas upon facts of his own studies, he had been influenced by the work of Adolf Meyer, Freund, Bleuler, and Jung.

Every person has certain points on which he is especially sensitive. He has ideas, or complexes of ideas, which are associated with very strong feelings. These complexes refer either to personal defects, shortcomings, limitations, or to feelings of guilt, remorse, shame; on the other hand to certain longings and desires. We may therefore, generally speaking, say that they belong either to the realm of self-assertion or to the sexual sphere, in the broadest sense of the term. Now most people are able to get square with such things, partly because their nature is such that these feelings never reach anything like a great intensity, or partly also because they have a healthy way of dealing with these matters.

Other people do *not* get square with such difficulties. They do not acquire balancing, healthy habits, such as a healthy turning away from one's difficulties to outside interests, or a habit of unburdening, or a certain aggressiveness, and the like. While, then, such undercurrents, as we may call these complexes, when they are of any intensity, have themselves a tendency to set narrower and narrower limits to the interest, and to create a certain fascination, they often become a menace to the sanity of mind, also because they are not balanced sufficiently by sound mental tendencies. In this way there develops a growing disharmony which gradually, or sometimes under the influence of acute causes, physical or mental, may suddenly lead to an unbalancing of the mind when, finally, the undercurrents break through to the surface.

But the mind, even in the cases in which the undercurrents are not handled properly, makes certain miscarried attempts at readjustment. Thus, the feelings of defect and the longings do

not come to the surface as such, but are transformed; the former give rise to a general suspiciousness and delusions of persecution, probably for the same reason that we are inclined to blame everyone else except ourselves when anything which we do goes wrong; the latter give rise to ideas that the innermost longings are fulfilled. And there are still other forms of such miscarried adjustments.

We see, then, that we have two things, the undercurrents and the abnormal manner of dealing with these undercurrents, upon which we should lay stress as important in the causation of these paranoic states. To a certain extent this division is of course artificial, and the two principles often enough overlap greatly. Then again it is difficult often to find a correct or a definite formula for that which we have called abnormal mental habits, or difficult to pick out from among the complex fabric of mental reactions those which are disastrous, or estimate the dangers of certain combinations, or to correctly gauge the value of saving traits. Naturally it will often be a combination of traits rather than single traits which we have to consider, and while we speak of some reactions as dangerous mental habits, they may exist in certain combinations in which they are sufficiently safeguarded.

It is also very evident that other causes than an unhealthy manner of dealing with the undercurrents may enter into the causal constellation as well—such as influences which increase the strength of the undercurrents or influences, which, in other ways than those indicated, lessen the resistance, such as the action of alcohol, the menopause, and the like.

These principles were demonstrated by means of careful analysis of four cases and certain indications for treatment were discussed.

Discussion.

Dr H. R. Stedman, of Boston, was inclined to lay more stress on the influence of heredity in affecting the progress of a genuine paranoia than did Dr Hoch. Numbers of cases of the disorder were seen in patients who had been sensibly brought up, and who were treated affectionately by their families, nothing being left undone to make their surroundings congenial and their lives smooth and happy, yet in spite of it all they developed paranoia. Little could be hoped for, he believed, in the way of materially modifying the psycho-genetic factors so as to make any real impression on these cases of typical paranoia, a disease arising on a defective constitutional basis, and gradually and logically developing into an inflexible system of delusional thought and conduct.

He thought, however, that after the disease had developed, when family, friends, and a normal environment had proved powerless to influence the disease, and the patient was sent to the hospital, his condition was more susceptible of improvement than is generally thought to be possible. He had not infrequently found the paranoiac to be rendered decidedly more manageable and his life made more comfortable by regular friendly and ex planatory talks, answering his questions, making the endeavour to set him right, and satisfying such of his minor demands as were not wholly unreasonable. The fact that many of them are hopeless and cannot be reached at all by such means—in fact only become worse in consequence—accounted, he thought, for the tendency that exists to pay them as a class little or no systematic attention, such as Dr Hoch adopts with his cases. Dr Stedman questioned if the reader had not chiefly in mind the paranoid state rather than the paranoic, that symptomatic, persecutory condition so often found in dementia præcox. If so, he was wholly in accord with his view that much might be done in the way of prophylaxis. Dr Hoch's masterly analysis of the psycho-genetic conditions in his cases showed this plainly, and he believed it to be due to the fact that the morbid direction of their thought had become less impaired than in the true paranoiac. Dr Stedman felt the same confidence that he had expressed at length several years ago, that not a few cases of this kind, when recognised early by the psychiatrist while yet the patient is comparatively comfortable, may be saved from an attack by well-directed medical oversight and guidance and regulation of his habits and surroundings. He attached little importance to the menopause as a special causative factor in insanity, as individual experience and statistics seem to show quite conclusively that paranoia develops to the same extent in both sexes during the period of life in which the menopause occurs.

Dr Charles L. Dana had been interested in Dr Hoch's analysis, which was instructive as showing that, in a certain group of cases of paranoia, conditions might be improved by careful therapeutic effort. He had not been in a position to carry out this method of treatment, which could not be very successfully employed by those not connected with institutions. He agreed with Dr Stedman as to the importance of hereditary taint in all these cases, and that a goodly proportion of paranoiacs develop in spite of careful bringing up. Few of these patients could be influenced unless they were taken in hand very early. He had been much interested in two or three cases of paranoia, which illustrated that the undercurrent does not always break through in a way that particularly disturbs the mental make-up or general life of the patient. Such a case was a woman, about fifty years of age, now under his care, who

was first seen by him when she was forty years old. She was married and the mother of two healthy children. About fifteen years before he first saw her she had developed delusions of a certain kind of persecution—that when she went out on the streets people made remarks about her, trying to annoy her and to injure her. She had these delusions throughout her married life and during her pregnancies. She was a good mother, however, and to most people who knew her she remained a good, kindly woman, about whose mental condition no one had suspicions except her husband, some members of her family, and Dr Dana. She was probably preserved from a general paranoic state by the fact that she was able to stay in the house and keep away from sources of irritation. He had had under observation also a man, now forty years old, who had been engaged in business all his life. For fifteen or twenty years this patient had had similar delusions of persecution—that the police and detectives were after him, and that attempts were being made to watch him. But this undercurrent delusion never broke through except in one little spot in his brain. One or two of his children developed dementia præcox at the age of sixteen. Such very limited types of paranoia certainly lent themselves to treatment by instruction and by careful selection of environment, which was all-essential. As to the general correctness of Dr Hoch's analysis there could be no question.

Dr Maurice C. Ashley, of Middletown, N.Y., agreed with Dr Hoch in the main, but he questioned whether the therapeutic talks with paranoiacs would accomplish very much as a curative measure. In his experience there had been no such beneficial results. He recalled one paranoiac who for ten years had believed that he had been giving him poison. At first the patient was inclined to retaliate; he threatened, and made definite efforts to take the life of the doctor's children. The man had some somatic symptoms, which he himself attributed to the poison which he thought had been given him. He still has the delusions, but no longer attempts to execute his threats. Another patient, a woman, for eight years had believed that he had been turning an electric current upon her for the purpose of annoying her. Every argument had been used to convince her that this was impossible, but without effect. As the disease progresses the reason of such patients becomes enfeebled and less active, and while they continue to have their delusions, they become accustomed to them and cease to react much to them.

Dr William Hirsch thought that in forming a definite opinion concerning the cases analysed by Dr Hoch, it must first be determined whether one had to deal with genuine paranoia, or with a paranoical state of another disease. Genuine paranoia is always

a congenital and not an acquired disease, although the true paranoical symptoms often do not manifest themselves during the earlier part of life. But there is always a congenital condition, a constellation of mental factors, which not only predisposes to, but which necessarily develops, at some time of life, such a combination as to produce that mental condition known as paranoia. When such a point in any given case would be reached cannot be determined in advance, but we are, in most cases, able to predict the development of a true paranoia. Various conditions, such as environment, worry, etc., might have something to do with it, at least with a premature manifestation of the condition. He did not believe, however, that in any given case anything could be done to prevent the manifestation of the paranoical condition, even though it were recognised that the development of such a condition existed. This opinion was not based merely on theory. In his practice he had had children brought to him whose parents realised that they were a little peculiar, nothing more, but whom he recognised as abnormal individuals who in later life would become paranoiacs. In such of these cases as he had been able to follow for ten or fifteen years, he had found that they developed genuine paranoia in spite of all the precautions which had been taken. He had warned the mother not to let the child have any impressions which would stimulate the imagination or fancy of the child, not to let it read any fiction, to guard it against any undue emotions; all this was carried out with the greatest care. But at some time in life—generally after an unusual emotion, such as falling in love, slight business troubles—something which otherwise would be of no importance—they would develop a true paranoia. A normal individual, normal from the start, would never develop paranoia. A normal individual might develop melancholia, or some other acute disease, but never paranoia. When he said one must differentiate between types, he meant cases in which there was genuine paranoia and those in which there was a paranoical state. The paranoical state might occur in a great many psychoses. He had seen such a case lately. A man of sixty years of age, a good business man, perfectly normal all his life, suddenly developed a paranoical condition; he had delusions and hallucinations, imagined there was a conspiracy against him, that his neighbours tried to kill him, etc. After remaining in this condition for nine months he gradually became demented. He is still living, and is suffering from a condition of general arterio-sclerosis. The case could be defined as dementia senilis, but not as paranoia.

Dr P. F. Knapp, of Boston, thought it a mistake always to regard delusions of persecution, with hallucinations of one form or another, as constituting paranoia, and that we should be guarded

in speaking of such conditions as paranoic states. He agreed entirely with Dr Hirsch's opinion that true paranoia, while not a congenital condition, is dependent upon a congenital mal-arrangement, so to speak, of the brain. Tanzi had taken the same position, viz., that whereas other forms of mental disease might be spoken of as true diseases, paranoia was not a disease but a morbid congenital state, which, later in life, under the influence of various factors, might develop into typical paranoia with hallucinations and delusions. He thought that the "undercurrent" did not always "break through." In this connection he cited the case of a woman who for years had had a limited type of delusion. She had lived a secluded, narrow life in one of the smaller New England cities; for many years she had been active in the care of her household and family and in church work; she had been trained in the old New England habit of keen theological discussion and argument, and for many years she had had the very definite idea that she had been excommunicated from the church. In the main the idea had been suppressed; many of her church associates did not know of it, and those who did kept it secret. The idea existed for many years without going on to any real mental disturbance. Cases were not uncommon in which the delusions occupied a limited field in the consciousness, and affected but little the conduct. With a true paranoiac, however, he questioned very much the real importance of any emotional stress, or of any psychical ideas as influencing materially the genesis of the disorder. They might influence the development in so far as changes in modern belief influence the character of delusions. As Dr Hirsch had suggested, it was impossible to protect these patients from all influences that might give rise to the condition. Not infrequently delusions of persecution developed in normal individuals in connection with hallucinatory conditions having a distinctly physical basis. He had recently seen such a case—a man with well-systematised delusions on an alcoholic basis, derived largely from tactile disturbances, which proved to arise from the paræsthesiæ of a very mild alcoholic neuritis.

Dr L. Pierce Clark was of the opinion that the cases cited by Dr Hoch might be called paranoid states rather than typical or true paranoia. The therapeutic suggestions outlined would be of undoubted value in these paranoid states. During the past three years he had been treating several cases by analyses and talks, and the method had been very advantageous. He thought the method was of little use in true paranoia, as the mental state was too fixed; his experience in asylum service had proved this fact to his entire satisfaction.

Dr Swepson J. Brooks, of Harrison, N.Y., was very glad to

know of the success Dr Hoch had had with therapeutic talks. He had tried this plan, and found it productive of results in many cases, but the patients would relapse into the old condition after being released from institutions. He presumed that Dr Hoch had reference in his paper to simple paranoid states. The question of paranoia was a hard one to go into, and sometimes one almost concluded that paranoia and paranoid states were the same, only differing in degree. The forcing of patients to do things, as suggested by Dr Hoch, was often neglected. He had in mind two cases in which it certainly had a very salutary effect. One case was a woman, forty-five years of age, who had delusions of persecution. She was put in a very quiet hall. She complained that she was merely brought to the place to be put in jail, that there were no sick people there, and that she would like to see some sick people. She was allowed to see some sick patients; the next morning she was convinced, and she got well. That was four years ago, and she had remained well since. The other case was of the manic-depressive type. The patient confessed after her recovery that her family physician had had to force her to take medicine, that he would stand her up against the wall and knock her head against it if she did not take the medicine, and that she believed his method did good.

Dr Smith Ely Jelliffe said that Dr Hoch's paper had offered glimpses into a large and but partly explored territory. To him, four different trends of thought were suggested, all of which were the subjects of much investigation. In the first place, the importance of the study of the mental development of the child was emphasised. The work of Weygandt on Abnormal Children, of Koch on Pathological Inferiority, of Hall in his masterly work on Adolescence, and of Sommer on Character and Personality were instances in point as to the activity of these lines of investigation. As to the psychogenic origin of certain types of delusions, Dr Jelliffe was in accord with Dr Hoch. He spoke of the help that might come from the literary side, as evidenced by the stories of Henry James, "The Turning of the Screw" and "The Two Magics"; Wier Mitchell's "Constance Trescott"; and Ansty's "Statement of Stella Maberly." In all these this type of delusion formation is beautifully brought out, with great literary charm, if not with scientific pedantry. Therapeutically he deemed Dr Hoch's paper as stimulating, and he himself regarded certain phases of the subject with optimism. Paranoia, he said, was too large a term to use in a general blanket manner. While it is true that little can be accomplished by the most tactful of psychotherapeutic conversations in chronic lunatics who have been in the asylums for years, yet the important factor in the whole problem is to recognise the beginning stages, before the delusional

ideas have become too firmly crystallised. Greater success had not been attained because the psychogenic origin of many delusional states had not been sufficiently understood. It required a rare tact to work on these patients, and the outlines given by Dubois, Déjerine, and Oppenheim were but the beginnings of a scientific psychotherapy which for some time has been grasped at by pseudo-scientists. Dr Jelliffe desired to rank himself with those who saw a hopeful outlook for the amelioration, if not cure, of certain cases of dementia præcox, and of the paranoid states, by early and intelligent psychotherapy.

Dr George H. Kirby had been interested of late in the management of paranoic states along the lines suggested by Dr Hoch, and thought that much could be accomplished in this way toward the correction of morbid trends. Dr Hoch's work was particularly important in regard to the study of delusions in general. Such a method of analysis opened the way to an understanding of certain mechanisms which heretofore had been practically inaccessible.

Dr Hoch, in closing the discussion, stated once more that what he wished to bring out was the fact that certain paranoic states were produced by purely mental causes, *i.e.* by conflicts and unhygienic ways of dealing with them, and that they were more or less amenable to treatment early in the course, but that naturally he did not mean to claim that old cases of paranoia could thus be influenced. It was necessary in order to help such cases that one could still get at the root of things, and explain to the patient the genesis of his delusions, and train him to healthy mental habits. The criticism that his cases were not cases of typical paranoia, he could not quite understand, because he was unable to see where the line could be drawn between cases such as his and cases of so-called typical paranoia. Again, to say that paranoia was caused by heredity was an exceedingly unsatisfactory way of stating the situation, because it did not mean enough. He had claimed that some paranoic states were due to an unhealthy dealing with conflicts. Such an unhealthy dealing may be due to tendencies which were more or less inherited, but that it was time to make an attempt at determining what these tendencies were, because the mere statement of heredity was absolutely barren, that the same may be said about the statement which has been made that paranoia was due to a congenital mal-arrangement. If Dr Hirsch said that a normal individual would not develop paranoia, this was doubtless true if by normal individual was meant one who had perfectly healthy mental habits.

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Review
of
Neurology and Psychiatry

Original Articles

THE PATHOLOGY OF EPIDEMIC CEREBRO-SPINAL MENINGITIS.[1]

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THE presence of Epidemic Cerebro-spinal Meningitis in Edinburgh and district during the past year has afforded me the opportunity of studying the disease from its pathological aspects. In my capacity of Pathologist to the Sick Children's Hospital I saw many of the earlier cases, and, through the courtesy of the authorities at the Edinburgh and Leith City Fever Hospitals, I was enabled to see or assist in several of the post-mortem examinations held in these institutions. Several practitioners have also kindly placed at my disposal material from private cases. I have thus been enabled to study over 50 cases, and, as the mortality has been exceptionally high, I have been enabled to perform over 30 post-mortem examinations. The following paper embodies some of the results obtained in this investigation. Special points in the bacteriology and observations on the

[1] A portion of a Thesis submitted for the degree of M.D. (Edin.) in April 1907, and awarded a Gold Medal. Towards the expenses of the investigation a Grant of £23 was received from the Moray Fund. The clinical aspects of Epidemic Cerebro-spinal Meningitis were dealt with by Dr J. Stewart Fowler in the April number of this Journal.

experimental production of the disease will be published in an early number of the *Journal of Pathology and Bacteriology*. The work has been mainly carried out in the Laboratory of the Royal College of Physicians, and I am much indebted to that Institution for the facilities afforded me there.

I propose in this number of the Journal to give a brief résumé of previous work on the subject; to describe some illustrative post-mortem examinations and to summarise the morbid anatomy and histology of the disease as I have personally observed it. In the next number of the Journal, some points in the bacteriology of the condition, the relation of the disease to Posterior Basal Meningitis, and the mode and paths of infection will be dealt with.

Historical Résumé of Work on the Etiology of Meningococcal Meningitis.

Though cerebro-spinal fever has been known as a clinical entity since 1805, when it was first recognised as a definite disease by Vieussieu in Geneva, and though accounts of post-mortem examinations are on record as early as 1809, when a committee, appointed by the Massachusetts Medical Society, reported on an outbreak of the disease, exact knowledge as to the bacteriology of the disease is of very recent date. In 1887, Weichselbaum (in Vienna) was the first to describe the micro-organism which has since become universally recognised as the specific cause of the disease. In his original communication (1), which dealt with an investigation into the bacteriology of eight cases of acute meningitis, he described two different organisms which he had found. In two of the cases the organism was an undoubted pneumococcus, but in the other cases the organisms present were of an entirely different nature. This second organism he described as occurring as a diplococcus, the opposed surfaces of the cocci being flattened. The organism was found chiefly inside the cells of the inflammatory exudate, though free forms were also seen. The organisms were decolourised in Gram's method, and their general morphology in the tissues closely resembled that of the gonococcus. He was able to cultivate the organism from the meningeal exudate, and was able to study its morphology and biology. He also recorded

some experimental observations on mice, guinea-pigs, rabbits, and dogs. Subcutaneous injection of cultures of the organism he found to have practically no effect, but fatal results were produced by intra-peritoneal and intra-pleural injections in mice and guinea-pigs. Sub-dural inoculation in one dog produced a purulent meningitis, with death on the third day. Weichselbaum at first regarded those six cases as examples of sporadic cerebro-spinal fever, but it was afterwards discovered that some of the cases at least had occurred in Vienna when the disease was epidemic there. To the organism which he described, Weichselbaum gave the name Diplococcus Intracellularis Meningitidis. The organism has since then become more familiarly known as the meningococcus.

Weichselbaum's discovery was confirmed by Goldschmidt and Edler in the following year. Both of those observers described cases, and their results practically agree with those of Weichselbaum.

In 1889 (2), and in 1892 (3), Netter gave the results of his researches on suppurative meningitis, and in 1890 (4), in a communication on the pneumococcus, he was evidently of the opinion that this was the organism specially concerned in the production of epidemic cerebro-spinal meningitis.

In 1894 (5), Flexner and Barker reported on an epidemic of meningitis which prevailed in the George's Creek district, Maryland, in 1893. In this outbreak, which included about 200 cases, they noted no special seasonal incidence of the disease. The insanitary conditions in the affected district were evidently very marked. From a study of a few cases they came to the conclusion that the causal organism was the pneumococcus. They gave an account of the morbid anatomy and histology of their cases, but their bacteriological results are not convincing. This is, partly at least, to be accounted for by the distance of the scene of their operations from a bacteriological laboratory, and the consequent delay in the incubation of their cultures and in their animal experiments. They thought it probable that the route of infection in many of the cases was by the intestinal tract, and they were of opinion that some general depreciation in the health of the community had a good deal to do with the outbreak of the disease. In 1895, Jäger (6), a German army surgeon, described some cases he had observed,

and while his results in the main confirmed those of Weichselbaum, he indicated some differences in the organisms he separated, from the classical description given by Weichselbaum. Jäger appears to be the first to definitely associate the epidemic disease with the meningococcus. Other points to which he drew special attention were: first, that the organism in culture sometimes occurred in chains of considerable length; secondly, that the organism in culture was Gram-positive, that is to say, retained the stain in Gram's method; and thirdly, that the organism was sometimes observed to be capsulated.

In 1896, Heubner (7) confirmed Jäger's observations, specially with reference to the Gram-positive nature of the organism. Heubner produced a typical meningitis in a goat with a culture of one of these Gram-positive organisms.

The following years were remarkable for the fierce controversy which arose between the followers of Weichselbaum and those of Jäger. Undoubtedly much loose writing prevailed about this period, and as Rist (8) remarked at a later date, "every coccus found upon the meninges about this time became identified with the meningococcus."

Papers by Kiefer (9), Kister (10), Kischensky (11), Fronz (12) confirmed Weichselbaum in his original description, but Mayer (13), Pfaundler (14), and Fränkel (15) described types of the meningococcus which were distinctly Gram-positive, or took, as several described, a "Mittelstellung" with regard to Gram's stain, that is to say, were sometimes Gram-positive and sometimes Gram-negative. Though the meningococcus was now more generally recognised as an essential cause of epidemic meningitis, numerous observers still ascribed an important rôle to the pneumococcus in the causation of the disease.

One of the most important papers which has appeared was published in 1898, when Councilman, Mallory, and Wright (16) reported on an outbreak of the disease in Boston, especially in 1897. In 35 cases, on which post-mortem examinations were made, meningococci were found in cultures or on microscopical examination of the exudate, or in microscopic sections of the tissues, in all but four cases. They gave a good description of the organism, which practically corresponds to Weichselbaum's. Mixed infections, with other organisms, were not uncommon: thus, the pneumococcus was found seven times in connection

with meningococcus, and once Friedländer's bacillus was present. Terminal infections, with streptococci and staphylococci, were occasionally seen. The organism they note as possessing only feeble pathogenic power towards the lower animals; in a small percentage of cases death was produced in guinea-pigs and rabbits by intra-pleural and intra-peritoneal inoculations. A typical meningitis was produced in a goat by inoculating directly into the spinal canal. In their communication a good account is given of the morbid anatomy and histology of the disease. Special reference is made to the minute changes produced in the central nervous system. The intestinal canal was found normal in every case. Lesions of the skin were found in but one of the cases in which post-mortem examination was made. In seven of the 35 cases there was broncho-pneumonia; in two cases there was croupous pneumonia. In these cases pneumococci were demonstrated in the lesions. Eight of the pneumonia cases they describe as definitely due to the meningococcus.

In the same year, 1898, Still (22) described the results of his research on posterior basal meningitis. This disease, which is not so uncommon in infants below two years of age, and occurs specially in the spring months of the year, was first described in 1878 by Drs Gee and Barlow (23). In practically all his cases, Still was able to demonstrate an organism very closely resembling the meningococcus. He was able to obtain cultures, and performed a considerable number of animal experiments. Though so closely resembling the meningococcus, Still indicated some differences, the most important of which was the greater longevity of the organism in culture. His work will be referred to again later.

During the last nine years numerous other papers on the etiology of cerebro-spinal meningitis have appeared in German, French, English, and American literature, and I shall content myself at present with merely referring to the more important of them.

In 1901, Albrecht and Ghon (20) practically confirmed Weichselbaum's original observations, and controverted those of Jäger. In 1901, an important monograph, "Die Cerebro-spinalmeningitis als Heeresseuche," was published by Jäger. In this a copious bibliography is given, and a record of cases which Jäger himself had studied since his previous publication

is also included. In the chapter on Etiology, Jäger appears to have departed to some extent from his previous position. Thus, he here describes that he was mistaken with regard to the presence of a capsule to the organism. He does not now describe the long streptococcal form of the organism in culture, but he still maintains the "Mittelstellung" view in regard to the staining of the organism by Gram's method.

As late as 1903, Weichselbaum, in reviewing the literature of the subject, concluded that it was not possible to exclude the pneumococcus as the infective agent in some epidemics of cerebro-spinal meningitis. In his most recent pronouncement, however, Weichselbaum is apparently now of opinion that all extensive epidemics are really due to the meningococcus.

In an extensive epidemic in Portugal in 1902-3, Bettencourt and Franca (21) demonstrated the meningococcus in over 270 cases; only in three cases were organisms other than the meningococcus found. In the more recent American epidemics the meningococcus has been proved to be the causal organism, and the results of the American observers support Weichselbaum's original contentions.

In 1905, von Lingelsheim (17) reported fully on an extensive outbreak in Germany. In the full account which he gives of the meningococcus he is emphatic on the subject of the organism being Gram-negative, both in the tissues and in cultures. Strongly opposed to this view, however, are the opinions expressed by Vansteenberghe and Grysez (18) in January 1906. These observers describe an organism from a fulminant case of cerebro-spinal meningitis which corresponds to the meningococcus, except in that it was strongly Gram-positive. (A full account of experimental work with their organisms is given.) This paper I must also refer to again later.

In 1906 appeared the first important experimental work on monkeys. Flexner (19) showed that the clinical and pathological features of the disease in man could be reproduced in apes, and further showed that it was possible to protect monkeys from the results of inoculation with the meningococcus by anti-sera from apes immunised to the meningococcus by injection of cocci and exudates from the peritoneal cavity of infected guinea-pigs.

Von Lingelsheim (24) obtained similar results, and in his articles gave one of the most exhaustive accounts of the

meningococcus which has yet appeared. Lastly, in 1907, Flexner (25) has confirmed his earlier work, and has added much to our knowledge of the biology of the meningococcus.

Post-Mortem Records of Some Cases Personally Observed.

Case 1.—G. S., aged $1\frac{1}{12}$, admitted Royal Hospital for Sick Children, under Dr John Thomson, June 27, 1906; died June 27, 1906.

History.—Quite well up till yesterday. This morning was feverish and fretful. No cough. No digestive disturbance. Had a fit $1\frac{1}{2}$ hours before admission. Died a few minutes after admission. Total duration of illness about 14 hours.

Sectio, June 28.—Body is that of a well-nourished child. No hæmorrhages on surface.

Abdomen.—No peritonitis. Numerous small hæmorrhages over peritoneal aspect of intestines, and over omentum and mesentery.

Stomach not dilated; contained viscid, bile-stained mucus; mucosa is thick and velvety; no ulcers; some subperitoneal hæmorrhages and numerous small submucous petechiæ.

Small Intestine shows marked enteritis, with adherent mucus; patches of hæmorrhagic infiltration and great prominence of all the lymphoid tissue; the Peyer's patches are intensely swollen, and some are hyperæmic and show some superficial loss of substance. The appearances are best marked near cæcum.

Large Intestine.—Hæmorrhages as above, but not so numerous. Solitary glands very prominent, but no ulcers.

Mesenteric Glands are much enlarged; most are pale, swollen, and œdematous, some are hyperæmic. The enlargement of the glands is apparently quite recent. The retroperitoneal glands are also hyperæmic and swollen.

Spleen enlarged; acutely congested; Malpighian bodies very prominent and pale.

Kidneys cloudy, but not to an intense degree.

Suprarenal Bodies show nil abnormal.

Liver intensely cloudy, with some fatty change; no focal necrosis. There is recent passive congestion, and apparently some minute hæmorrhages at parts.

Thorax.—Some small hæmorrhages were present in the muscles of thoracic wall.

Thymus distinctly enlarged. Some petechiæ over lungs and a few over epicardium.

Heart shows nil beyond great pallor of muscle.

Lungs.—Some congestion and collapse at both bases, but no consolidation.

Brain.—Hyperæmia of convolutions, but no actual meningitis; great œdema of the brain.

Films were prepared from the fluid in the sub-arachnoid space over the vertex, and stained with carbol-thionin. The appearances were character-

istic of an early, acute, inflammatory condition. There were numerous well-preserved polymorphs, and a few large mono-nuclear cells. No organisms were discovered, however. Cultivations were attempted on agar and blood agar, but no growth was obtained.

This case I have described for several reasons. In the first place, though bacteriological proof of the real nature of the condition is wanting, I believe the case to have been one of fulminant cerebro-spinal meningitis, and further, that it is one of the first of the Edinburgh epidemic cases. The case must be read together with Case 2. A further point of interest is, that the case illustrates a variety of the disease which, for want of a better term, I shall designate the gastro-intestinal type, and though, for reasons to be afterwards adduced, the gastro-intestinal lesions may be regarded as secondary, it is possible that after all they constitute the primary lesion. The post-mortem diagnosis was acute general toxæmia from acute gastro-enteritis, but so impressed was I with the resemblance of the clinical features and morbid anatomy to fulminant cerebro-spinal meningitis that I suggested then the possible meningococcal nature of the case. Later, the same day, I heard that an older brother from the same house had been admitted to the Royal Infirmary with somewhat similar symptoms, and was gravely ill. I at once communicated with the pathologists of that institution, and informed them that I believed I had that day performed a sectio on a case of acute cerebro-spinal meningitis, though I had failed to find bacteriological evidence, and warned them, in the event of a fatal issue in the second case, to be on the outlook for a similar case.

Case 2.—J. S., æt. 2½ (an older brother of the previous patient), admitted to the Royal Infirmary on June 26, 1906. On the afternoon of the same date the child had fallen out of his perambulator, had been taken home, and was found to be distinctly ill. There was distinct pallor, and a series of slight convulsive attacks. He was admitted to the infirmary at 11.30 P.M. After admission there was constant diarrhœa, with green offensive motions. There were occasionally convulsive seizures, with stiffness, especially of the arms, neck, and head. There was distinct head retraction. The temperature varied from normal to 104°. Respirations were 70. The child died on June 28; the total duration of illness was about 48 hours.

Post-mortem Examination on June 29th, by Mr H. Wade. Rigor mortis was not present. There was well-marked post-mortem lividity on the posterior aspect of the body, and post-mortem discoloration of the abdomen. There

was a sub-cutaneous ecchymosis, about the size of a threepenny piece, on the dorsal aspect of the right wrist, but no other cutaneous hæmorrhages.

Serous Cavities.—The pleural cavities showed no fluid effusion and no adhesions.

Pericardial Sac contained about 1½ oz. of purulent fluid, yellow in colour, with a distinct green tint.

Heart.—The epicardium was covered with yellowish flakes of lymph, which were easily detached. The epicardium was very congested and showed some petechial hæmorrhages. The right auricle and tricuspid orifice were dilated. There were some stringy, ante-mortem thrombi in the right ventricle. The pulmonary, mitral, and aortic valves were healthy. The coronary arteries showed nothing abnormal. The myocardium was fairly firm and showed some pallor and slight cloudy swelling.

The *Thymus* was slightly enlarged.

The *Larynx* was congested; the hyperæmia was most marked over the aryteno-epiglottidean folds. The *Trachea* showed nothing abnormal.

Lungs.—The right bronchus contained purulent mucus. The right bronchial glands were intensely congested. A few petechial hæmorrhages were scattered over the visceral pleura, which was slightly congested and opaque. *On section* the right lung was acutely congested, but crepitant throughout.

Left Lung.—The bronchi showed nothing specially abnormal. Bronchial glands were much congested. The surface of the lung resembled the right. *On section* the organ was acutely congested.

Liver showed cloudy swelling.

Spleen.—Consistence somewhat soft; acute congestion.

Kidneys soft and pale.

Abdomen.—Mesenteric Glands were acutely enlarged. Some showed early caseation.

Intestines.—The jejunum showed numerous sub-serous hæmorrhages throughout its entire length. There was some catarrh about the root of the appendix.

Large Intestine in its lower part showed marked prominence of the solitary glands.

Both knee-joints contained purulent fluid.

Head.—On removing the dura, there was found a marked lepto-meningitis, with greenish-yellow purulent fluid in the sub-arachnoid space.

Bacteriology.—Films from the meninges, pericardial sac, and knee-joint showed numerous polymorpho-nuclear leucocytes, many containing typical meningococci. Cultures from those situations gave a copious pure growth of meningococci. The bacteriological investigation of the case was carried out by Dr W. T. Ritchie.

Although, as I have said, complete proof of the nature of the first case is wanting, there is strong presumptive evidence in favour of the view that the cases were of the same nature.

CASE 3.—J. G., æt. 4 years, admitted to Royal Hospital for Sick Children on February 9, 1907; died February 11. Illness started 3 A.M. on morning of admission, with fever, pain in head and back, and delirium. On

admission, patient had a slight measly rash. On the following day the stiffness of the back was very marked. Lumbar puncture was performed at frequent intervals, and numerous meningococci were found present, both free and intra-cellular forms being numerous; 6½ oz. of fluid in all were removed under considerable pressure. Death occurred at noon on February 11. Total duration of illness was about 57 hours.

Sectio, February 12, 1907, 24 *hours after death.*—Body well nourished. Rigor mortis general. Marked post-mortem lividity and mottling; some dark spots on chest, like small hæmorrhages in skin.

Brain.—Acute lepto-meningitis, basal and vertical; convolutions flattened; superficial vessels much engorged, with hyperæmic areas; exudate irregularly distributed; it has a distinct greenish tint, and is best marked along sulci. No evidence of spread from cribriform plate or temporal bone. All the nerves at the base were covered with exudate. Left mid-ear shows gelatinous, turbid-looking fluid. Left optic nerve and back of eyeball removed shows nil abnormal to naked eye.

Cord.—Some thick, fibrinous, gelatinous exudate outside dura over lower part of cord; acute lepto-meningitis of cord; upper cervical region shows very little; most exudate over lower part of cervical and lumbar enlargements. On section there is acute dilatation of central canal; in cervical and lumbar enlargement, especially in latter region, canal here on section fully $\frac{1}{16}$ in. across, and walls somewhat softened.

Neck.—No enlarged glands; nose and pharynx covered with muco-purulent matter; adenoids present.

Thorax.—Heart.—Myocardium cloudy; no pericarditis.

Lungs.—No pleurisy; emphysema; congestion and œdema; well-marked broncho-pneumonia in left lower lobe, with dilatation and congestion of small bronchioles.

Thymus not enlarged.

Liver cloudy and fatty.

Spleen enlarged slightly; acute congestion, with hæmorrhages in pulp. Malpighian bodies not prominent.

Kidneys.—Marked cloudy swelling.

Stomach.—Intestines and mesenteric glands show nil abnormal.

Bone Marrow of Ribs red and lymphoid.

Case 17.—P. W., æt. 3, a dispensary patient, seen by Drs Stewart Fowler and W. Macrae Taylor on February 28, 1907.

History.—The illness started about midnight on February 27, with vomiting, fever, and pain in the head. The vomiting continued for the following twenty-four hours. On examination, the child was irritable; there was a left internal squint, but no head retraction. Movement of the head or back caused evident pain. On lumbar puncture, fully 10 c.c. of a slightly opalescent fluid was obtained. It was not apparently under any great pressure.

Films showed many polymorpho-nuclear leucocytes, and typical meningococci; intracellular forms, however, were distinctly scanty. The case was

very acute, and the patient died thirty-eight hours after the onset of the illness.

Cultivations of the fluid were made on ovarian agar, and a copious pure growth of the meningococcus was obtained.

Unfortunately permission for a post-mortem examination was not obtained, but the clinical history and the examination of the cerebro-spinal fluid leave no doubt as to the nature of the case.

Though the details were so scanty, the case is important in relation to the following one.

CASE 18.—Baby W. The child, a baby of two weeks old (a sister of the previous patient's), became ill on Saturday, March 2. She was seen by the same dispensary student who had attended the older sister. The patient was a perfectly healthy infant, and had been breast-fed till the older sister was taken ill. From that time it had been bottle-fed. The symptoms were gastro-intestinal, and of no great severity. With her previous experience before her, the dispensary attendant was on the outlook for cerebro-spinal symptoms, but saw nothing at all suggestive of meningitis. Late in the evening the child became much worse, and died early the following morning. The total duration of illness was about eighteen hours. Through the courtesy of Dr Macrae Taylor I was afforded the opportunity of making a post-mortem examination, which was carried out on Monday, March 4, twenty-eight hours after death.

Sectio.—The body was that of a healthy-looking, well-nourished child. There was some distinct mottling of the skin at several places, but there was no hæmorrhagic rash.

Serous Cavities.—Pleuræ.—No fluid effusion, and no evidence of pleurisy. *Pericardial Sac* shows nil abnormal.

Lungs.—Bronchial glands show a slight degree of recent inflammatory change. The larger bronchi are congested, and contain frothy mucus. The surface of both lungs showed patches of lobular collapse and emphysema. *On section* there was well-marked broncho-pneumonia in both lower lobes, being specially marked on the right side.

Heart.—The epicardium showed nothing abnormal. The heart was flabby, and the right auricle and ventricle were dilated. There was no endocarditis. Myocardium was pale and showed some cloudy swelling.

Abdomen.—The mesenteric glands were distinctly enlarged, hyperæmic on the surface, and on section soft and succulent. The larger glands on section showed some pinkish hyperæmic areas. The *Stomach* showed nothing abnormal.

Small Intestine.—There was an appearance of early acute catarrh, the lymphoid tissue was prominent, and the Peyer's patches, towards the lower part of the small intestine, were distinctly swollen and slightly hyperæmic.

Large Intestine showed nothing abnormal.

Spleen.—The organ was slightly enlarged, firm in consistence, and dark red in colour. The Malpighian bodies were not unduly prominent. There were no hæmorrhages in the splenic pulp.

The *Liver* was cloudy and slightly fatty. The fatty change was more

marked in the peripheral zone of the hepatic lobules, and somewhat unequally distributed throughout the organ.

The *Kidneys* were pale and showed acute toxic change.

The *Pancreas* and *Suprarenal Glands* showed nothing abnormal.

The *Brain.*—The vessels of the pia-arachnoid were distinctly injected, but there was no excess of fluid or turbidity in the membrane. There was no exudate at the base of the brain. To the naked eye there was no appearance of meningitis.

Spinal Cord.—There was no trace of exudate on the cord, but the pia-arachnoid was very distinctly hyperæmic. The hyperæmia had no special distribution.

The impression formed from a naked-eye examination of the organs was that the child had died from an acute pneumonic infection, possibly pneumococcal in origin, with a secondary general toxæmia.

A bacteriological examination, however, revealed the real nature of the condition. On passing the loop of a platinum needle into the posterior arachnoid cistern, some fluid, faintly, yet distinctly turbid, was obtained.

Films were prepared from this in the usual way. On being stained these gave evidence of undoubted acute inflammatory condition. Numerous polymorphs and a few large mono-nuclear cells were seen, and a few diplococci, with the typical morphological appearances of meningococci, were found inside the polymorphs and also free. The organisms were distinctly Gram-negative.

Cultures were made on serum agar, but no growth was obtained. Cultivation was also attempted from the heart blood on serum agar, but the tubes remained sterile.

CASE 26.—Mrs W., æt. 39, taken ill on April 14, 1907, admitted to City Fever Hospital on April 15; died April 17.

Vomiting, shivering, headache (later convulsions). Head retracted. Knees drawn up. Abdominal reflexes absent. Not quite conscious. Can answer questions, but mentally confused. No fluid on lumbar puncture. April 16, much worse. Strabismus, vomiting, later convulsions and death. Quite conscious before the end. Total duration of illness, four days.

Sectio, April 19.—The examination was limited to examination of the brain and cord.

Brain.—On removing the dura, there was found no flattening of the convolutions. There was distinct hyperæmia of the surface of the brain, with marked injection of the minute superficial vessels, and a few extremely minute petechiæ, with no special distribution. There was practically no purulent exudate on any part of the brain surface, but the pia-arachnoid had a faint cloudiness along the sulci, evidently due to a small quantity of very slightly turbid fluid exudate in the meshes of the pia-arachnoid. On section, the brain was œdematous; there was no dilatation of the ventricles. There was practically no fluid in the spinal sub-dural space, but there was undue hyperæmia of the surface of the cord. There was no inflammatory exudate visible to the naked eye.

This case I have described, for though the clinical symptoms pointed to

acute cerebro-spinal meningitis, no fluid was obtained by lumbar puncture during life, and even at the post-mortem the naked-eye appearance of the brain and cord left one still in doubt as to the real nature of the case. Microscopical examination, however, confirmed the diagnosis.

Bacteriology.—Films from the fluid in the lateral ventricles showed a few inflammatory cells, but no distinct organisms. Examination of films from the fluid in the pia-arachnoid over the vertex gave a positive result. Numerous cells were present, some being polymorphs, the majority, however, being large mono-nuclears. There was active phagocytosis on the part of those latter cells towards the polymorphs and degenerate red cells, together with vacuolation of the phagocytes. Both free and inside the cells were numerous faintly and irregularly staining particles, representing apparently degenerating diplococci. Only after a prolonged search were typical meningococci found. In the light of such an experience it is possible that a more prolonged search in the films from Case 1 would have given an equally satisfactory result.

Case 27.—David B., æt. 11 months, taken ill January 13, 1907, admitted to Sick Children's Hospital on January 16; died April 18, 1907.

On lumbar puncture, turbid fluid showed typical meningococci. Vomiting and head retraction. Quite conscious. Improved slightly during first fortnight after admission. Temperature averaged 101° with marked variations. Later, began wasting. Head retraction extreme. Opisthotonos. Heels approximated to buttocks. Slowly sank and died in this condition, extraordinarily wasted, 14 weeks from admission.

Sectio, April 19, 1907.—Rigor mortis was present, but slight. There was marked emaciation. There was still present some head retraction and backward curving of the spine. There were no cutaneous hæmorrhages.

Serous Cavities.—No fluid effusion and no adhesions in pleural or pericardial sacs.

Thymus not enlarged.

Lungs.—No enlargement of bronchial glands. Some bronchitis in the larger air passages. There was well-marked emphysema on both sides, with patches of lobular collapse and early broncho-pneumonia.

Heart.—Beyond some dilatation of the right side of the heart, nothing abnormal was noticed.

Abdomen.—*Mesenteric Glands* slightly enlarged, pale and firm, no evidence of tubercle.

Stomach and *Intestines* showed practically nothing abnormal.

Liver.—Early chronic venous congestion, with some fatty change.

Spleen not enlarged, pale and firm. Malpighian bodies hardly apparent to naked eye.

Suprarenal Bodies showed nothing abnormal.

Kidneys.—Nothing abnormal, beyond distinct pallor.

Brain.—There was well-marked but resolving lepto-meningitis at the base of the brain, and extending upwards along the sulci to the extreme vertex on both sides. There was still thick, semi-solid, gelatinous yellow lymph in the posterior arachnoid cistern, and scattered over the pia-arach-

noid, both at the base of the brain and over the vertex, were numerous yellowish-white particles of resolving inspissated lymph. These were very distinctly situated on, and adherent to, the inner aspect of the pia-arachnoid. They were well seen also on the membrane bridging the Sylvian fissures. They might readily have been mistaken for tubercles by anyone unfamiliar with the appearances of a resolving meningitis.

Cord was practically free from exudate, and there were remarkably few adhesions between the dura and the pia-arachnoid.

The bone marrow of the ribs was scanty in amount, and pale red in colour.

Bacteriology.—Repeated attempts to cultivate the organisms from the cerebro-spinal fluid during life failed. This case is included to illustrate a chronic case. Clinically it corresponded completely to Posterior Basal Meningitis.

CASE 9.—Winifred M. C., admitted to Sick Children's Hospital, Dr Fowler, on February 13, 1907 ; died on February 14, 1907.

History.—Illness began with vomiting five days previous to admission. There was also pain and stiffness of neck. On admission, the child did not appear gravely ill. There was no head retraction, but there was pain on flexing the head forwards. Lumbar puncture yielded fully 5 c.c. of thin, slightly turbid, blood-stained fluid. Films showed comparatively few cells, mostly polymorphs, many degenerating. Numerous diplococci were present. Many were intra-cellular, but enormous numbers were free. A few of the intra-cellular forms were somewhat rounded, slightly suggesting meningococci, but most, including all the extra-cellular forms, were distinctly elongated, resembling pneumococci. They were distinctly capsulated. Cultures on agar and blood agar gave in twenty hours a copious pure growth of pneumococci.

The patient became suddenly worse about midnight, February 13. Signs of pneumonia appeared at both bases. The pulse became uncountable and she died at 2 A.M. the following morning.

Sectio, February 15.—Permission was granted for examination of head and spine only.

Brain.—On removing the dura there was found a diffuse suppurative lepto-meningitis. The exudate at the base was scanty. The surface of medulla and posterior arachnoid cistern were free from exudate. The superficial vessels over the vertex were markedly injected, and over the whole of the vertex, but especially anteriorly, there was a thick layer of greenish-yellow gelatinous pus covering the convolutions in a uniform sheet. The exudate was present not only in the meshes of the pia-arachnoid, but also on its surface, and much of it could be readily removed. There was no evidence of a spread of infection from either the nose or the ear.

Cord.—The surface of the cord was markedly injected, but there was very little free fluid present, and practically no solid exudate.

Films from the exudate showed numerous diplococci, all pointed and capsulated ; none resembled meningococci. Inoculation of the cerebro-spinal fluid from this case into a mouse confirmed the diagnosis of pneumococcus.

This case has been included to illustrate the point that, in the course of an epidemic of cerebro-spinal meningitis, pneumococcal cases occur which can only be recognised by a bacteriological examination.

The post-mortem record of another acute meningococcal case was published in the *Scottish Medical and Surgical Journal*, March 1907.

THE MORBID ANATOMY OF MENINGOCOCCAL MENINGITIS, BASED ON THE STUDY OF THE RECORDS OF THIRTY-THREE POST-MORTEM EXAMINATIONS.

External Appearances of the Body.—The state of general nutrition of the body has varied considerably: some of the acute cases have obviously occurred in poorly nourished children, but the good development and healthy state of nutrition in the body generally has been quite a marked feature on many occasions. In the more chronic cases, marked emaciation has been a very striking character.

At the time of post-mortem, in the few cases in which a hæmorrhagic eruption had been present during life, the petechiæ have been little prominent, but in many cases there has been quite a distinct mottling or blotching of the skin. In one case subconjunctival hæmorrhages were seen.

Buccal Cavity.—In many cases the mouth has been noted as being particularly foul, and there has often been seen a copious viscid, muco-purulent secretion about the tonsils, palate, and back of throat. A similar secretion has often been noted in the nose and naso-pharynx, but the nasal cavities have been often observed in an apparently healthy condition. A muco-purulent conjunctivitis has been noted at the sectio on two conditions; adenoids have also been occasionally seen.

Thorax.—Serous Cavities.—Fluid effusion into the pleural cavities has been quite the exception. In the cases where there has been extensive pneumonic consolidation, a small amount of fluid has been present, together with small flakes of lymph on the visceral pleura. There has been little exudation also into the pericardial sac. In two cases, however, there was a purulent pericarditis.

Heart.—Minute petechiæ have been seen below the epicardium in a few of the acute cases.

The organ as a whole has in the majority of cases been soft, flabby, and dilated. No endocarditis has been noted. The myocardium, except in the more chronic cases, has invariably showed an acute degree of cloudy swelling, with small areas of hyperæmia in the substance of the muscle.

Lungs.—On the visceral pleura, minute petechiæ have not been uncommon in the acute cases. The bronchial glands have shown no special change; in one case they were definitely tuberculous.

The bronchi have been usually congested, and showed muco-purulent exudate on the mucous surface. Acute congestion and œdema of the organ has been almost invariably present, and in some cases has been extreme. Pneumonia of broncho-pneumonic type has been present in over 65 per cent. of the cases; in only one case was there tuberculosis in the substance of the lungs.

Neck.—The lymphatic glands in the neck have shown no special enlargement. The thyroid gland has shown no special change except in one case where there was a congenital goitre. The thymus gland has been enlarged, soft, and succulent in quite a number of cases.

Abdomen.—Peritonitis has not been noted, but in a few cases there have been small sub-peritoneal hæmorrhages.

The Stomach has shown no special abnormality, except in Case 1. The intestines in many cases have shown no special change, but in one case there was a hæmorrhagic enteritis, and in the majority of cases there have been slight degrees of a simple inflammatory change. The lymphoid tissue in the Peyer's patches and solitary glands has frequently been noted as being unduly prominent. The mesenteric glands have almost invariably shown some change. In a few cases they were obviously tubercular. In some they were slightly enlarged, but pale and firm. In a few, as, for example, in the case of Baby W., Case 18, they were obviously recently enlarged, and acutely inflamed. (The total duration of this case, it may be noted in passing, was less than 24 hours.) In some cases this recent enlargement reached a diameter of from ½ to ¾ of an inch, and on section those glands showed areas of hyperæmia or actual hæmorrhage. In a few cases the retro-peritoneal glands showed a similar enlargement, but of lesser degree.

Suprarenal Bodies.—In a few cases, notably in the case of Baby W. just referred to, there was extensive hæmorrhage into the substance of the gland.

Kidneys.—In practically every case there was evidence of acute toxic change, and, in the more chronic cases, areas

indicating fatty degeneration were present. In one case there were multiple infarcts, evidently pyæmic in nature, in one kidney, but in no case was there present hæmorrhagic nephritis, such as has been described by Councilman (26) and others, and such as I have recently seen in a case of Professor Greenfield's.

The Ureters, bladder and pelvic organs have shown no special abnormality.

The Spleen.—The organ has been practically always slightly enlarged, fairly firm in consistence, and on section showing undue prominence of the Malpighian bodies, with areas of hyperæmia or actual hæmorrhage in the splenic pulp.

The Liver.—The organ has shown practically nothing characteristic; there has been almost invariably acute toxic, and in some instances fatty change.

Suppurative arthritis was noted in the knee-joints in two cases.

Cranial Sinuses, etc.—In a few cases the sphenoidal and ethmoidal sinuses have contained muco-purulent contents, but in many cases they have shown practically nothing abnormal. The appearances have given me the impression that the changes are secondary, due to a spread of the disease from within. The middle ears, especially in the younger patients, have often been noted to contain gelatinous or muco-purulent contents. This, however, in my experience, is a very common appearance in children dying from various general diseases.

The central nervous system shows practically the only constant features characteristic of this disease.

The Brain.—The dura mater has shown no striking change. On removing it, the surface of the brain has been found in all cases, with the possible exception of B., Case 27 (which lasted fourteen weeks), to be markedly hyperæmic, with an exudate in the meshes of the pia-arachnoid varying from a very slightly turbid fluid to thick, gelatinous, yellow lymph. In nearly every case the exudate on the sides and vertex of the brain has been pretty strictly limited to the sulci, but in a few cases it has shown a slight tendency to spread over the adjacent convolutions, and in one case it entirely covered the convolutions in a thick gelatinous sheet, giving an appearance indistinguishable from that seen in a typical case of pneumococcal meningitis. On removing the brain the exudate has been found almost invariably

to be most marked at the base posteriorly. In the typical acute cases the exudate has been gelatinous or definitely purulent; yellow in colour, with a faint greenish tint. A special site at which this exudate had accumulated has been the posterior arachnoid cistern, which has been frequently seen full of the exudate. From this region the exudate extends forward on the under surface of the medulla and pons as far as the optic chiasma, but it has been seldom seen to any extent anterior to this point. The exudate has often been noted also, extending both on the under and on the upper surface of the cerebellum near the middle line. In the very acute cases there has been some flattening of the convolutions, but in many of them there has been no distinct evidence of excessive intra-cranial tension.

The brain on section has been œdematous, and there has been sometimes purulent exudate in the lateral ventricles. In the more chronic cases there has been distinct hydrocephalus, with marked flattening of the convolutions and less hyperæmia of the surface of the brain. In the most chronic cases, too (such as that of B., Case 27), the exudate at the base and elsewhere may be largely removed, with an accompanying production of inflammatory adhesions. Minute particles of inspissated lymph are sometimes left in the walls of the posterior arachnoid cistern and in the pia-arachnoid bridging the Sylvian fissures and general sulci of the cortex. These, at first sight, might conceivably be mistaken for tubercles.

The nerve roots at the base of the brain in the acute cases are surrounded by purulent lymph.

Spinal Cord.—In a few cases an acute inflammatory exudation has been noted on the external surface of the dura. On slitting up the dura, in practically all cases there has been seen an acute leptomeningitis, and in many cases it has appeared to me that the lesions here are older than those seen on the surface of the brain. In the most acute cases there has been a considerable quantity of free fluid present, practically always turbid and sometimes blood-stained. In a few of the most acute cases there has been little noted beyond hyperæmia of the pia-arachnoid and a faint turbidity; but in the great majority of cases there has been a copious exudate of lymph similar to that seen at the base of the brain, sometimes covering the cord in a uniform sheet, sometimes more patchy in its distribution, and

always best marked on the posterior aspect of the cord. There has been less exudate in a great majority of the cases over the cord in the upper cervical region. The roots of the cauda equina have been matted with lymph in the acute cases, and a similar exudate has practically always been around the posterior nerve roots.

In the sub-acute and chronic cases there has been sometimes remarkably little exudate, but in most instances where the lymph has been absorbed, inflammatory adhesions have been left between the dura and the pia-arachnoid.

The Bone Marrow in the acute and sub-acute cases has been excessive in amount, and red and lymphoid in character. In the more chronic cases it has been paler red and watery.

The Histology of Meningococcal Meningitis.

Of the large amount of histological material now at my disposal I have been able to make little use so far, and I propose to deal with this branch of the subject very briefly at present. I have studied in detail the organs from ten of the cases, and the following observations are based on the microscopical appearances found.

The heart has in every instance shown a marked degree of cloudy swelling, with, in some cases, an œdematous separation of the muscle fibres and a degree of acute interstitial myocarditis.

The lungs have practically invariably shown acute congestion and œdema; in some cases this has been extreme, with much hæmorrhage into the alveoli; where pneumonia has been present, it has been of broncho-pneumonic type. In some cases collections of mononuclear cells of a lymphocyte type have been noted in the walls of the bronchi. With reference to the presence of meningococci in the lungs, it is difficult to make an absolutely positive statement; the degenerated organisms are so closely simulated by granular tissue debris. In some cases Gram-positive cocci and pneumococci were noted.

Spleen.—There was invariably acute hyperæmia present: a special feature was the proliferation of endothelial cells in the centres of the Malpighian bodies, and in the walls of the lymph sinuses. These cells were actively phagocytic towards

leucocytes, but not to great extent towards the red corpuscles. There was comparatively little blood pigment present. Hyaline degeneration of the arterioles was either very slight or altogether absent.

Suprarenals.—These showed acute parenchymatous degeneration, with intense hyperæmia, and in two cases actual hæmorrhages.

The Kidneys showed invariably very acute cloudy swelling. with nuclear degeneration. The epithelium lining Bowman's capsule showed no proliferative change; no organisms were demonstrated in the organ.

The Liver showed marked cloudy swelling in all cases, there was little fatty change present.

The Intestines showed in one case very distinct catarrh, and in several cases there was a marked lymphocytic infiltration in the sub-mucous coat.

The mesenteric glands were examined in four cases, where the enlargement was noted as being marked at the post-mortem. They all showed acute hyperæmia, with distension of the lymph sinuses, and active proliferation of endothelial cells. In one case, Baby W., Case 18, there were necrotic foci present, and I am of opinion that diplococci, resembling meningococci, are present. It is difficult to say, however, as only a very few well-preserved cocci were seen: the great majority were degenerated.

For purposes of comparison some acutely inflamed mesenteric glands from a case of acute pneumonia were examined. These were also hyperæmic, but did not show the same degree of endothelial proliferation that was observed in the meningococcal cases.

The Brain.—The exudate in nearly all the cases showed a great number, often a great preponderance, of large mononuclear cells apparently endothelial in origin. Some of these cells were observed in karyokinesis; they were very actively phagocytic towards the polymorphs; many were vacuolated and degenerating. Typical meningococci, both intracellular and free, were found in all but one of the acute cases; they appeared in some cases to be lying actually inside the nuclei. In the minute vessels of the meninges numerous leucocytes were present: nearly all were polymorphs or immature polymorphs; there was comparatively little fibrin present in the exudate. In none of

these cases so far has any acute endarteritis been noted, but the vessels at the base of the brain have not been specially examined. In the brain itself numerous isolated polymorphs were present; there was lymphocytic infiltration of the perivascular lymph spaces dipping into the cortex. There appeared to be some neuroglial proliferation in some cases. Some of the pyramidal cortical cells appeared to show acute degenerative changes, but the appearances may be artificial. In the most chronic case (No. 25) there was very little exudate on the surface, but there were numerous fairly large vessels in the superficial cortex. In the pneumococcal case (No. 9) there was more fibrin in the exudate, fewer large mononuclear cells, and enormous numbers of pneumococci.

Spinal Cord.—The appearances of the exudate here completely correspond with those in the brain, and in every case there was marked cellular infiltration around the nerve roots. These latter appeared to show some acute degenerative change, and lymphocytic infiltration of their substance was also noted in some cases. There was also lymphocytic infiltration along the perivascular lymph spaces in the cord, and in one case there was a remarkable dilatation of the central canal. This, however, may be congenital and accidental.

I have not studied yet in detail the histology of the blood and bone marrow. A polymorphonuclear leucocytosis is a feature of the condition, as is also the large number of immature polymorphs which may be observed in the acute cases.

(*To be continued.*)

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A CASE OF NARCOLEPSY

By Sir WILLIAM R. GOWERS, M.D., F.R.S.

TRUE narcolepsy is sufficiently rare to justify the description of any well-marked case.

A girl, who was twenty-two when first seen, without neurotic heredity, had suffered, since the age of sixteen, from peculiar brief attacks of sleep. No cause for their commencement could be ascertained, except the scarcely adequate influence of early rising. One to three attacks usually occurred on successive days, and then an interval of one to two months elapsed before there were others. No exciting cause for them could be found. They occurred either in the morning or in the afternoon. Each lasted five minutes or longer, seldom a quarter of an hour.

An attack began with sudden yawning and great heaviness of the eyes, so that she found it impossible to keep them open; in a minute or two she was asleep and dreaming vividly. Sometimes the dreams were about familiar things or persons, sometimes about quite strange subjects, but beyond this general impression their details could never be recalled. Often she talked to herself during the sleep, and even seemed to rouse herself by doing so, waking in the middle of a sentence. She could generally be easily awakened by being spoken to, but she had to make a considerable effort to rouse herself completely, and often went off to sleep again a second time. If she at once yielded to the tendency to sleep when it was first felt, the sleep did not last more than about five minutes, but if she struggled against it, striving to keep awake, sleep always conquered her in a short time, and then lasted longer, ten or fifteen minutes. She was seen occasionally for a year, and had a few more prolonged attacks. In one, which lasted an hour, some darkness under the eyes was noticed, but otherwise her colour never changed during an attack. Once a series lasted most of the day. Her sleep at night was always good, and her general health was perfect. No headache followed the morbid sleep.

Various medicines were tried, but only one exerted an influence on the attacks, citrate of caffein. Under this she was free for several months, but they recurred when it was omitted and then its effect was slighter. When last seen, the attacks still occurred, although at longer ntervals.

This case is very similar to that for which the name "narcolepsy" was introduced by Gelineau in 1880.[1] His patient was a man of thirty-eight, whose sleep attacks commenced at thirty-six and lasted from one to five minutes. They occurred under any circumstances. There was never passage of urine or fæces. He is said to have had sometimes as many as 200 a day. Otherwise his condition was normal. No treatment had any influence upon the malady.

Gelineau quotes as similar a case described by Caffe in 1862, but in this the sleep was continuous. When roused the patient (a man of forty-seven) at once went to sleep again. Gelineau discusses the pathology of the affection, but his reasoning, by which he concluded that it is an affection of the pons, is scarcely worth consideration.

Cases have since been described under the same name which differ much in character. That of a boy aged eighteen, described by Foot[2] in 1887, was apparently similar to Gelineau's, but his somnolent attacks were said not to have involved loss of consciousness. A case described by Caton in 1889[3] was one of continuous sleep, except during active exercise. Continuous sleep has been called by the same name by other writers, but certainly should have another designation. The term "sleeping-sickness" has been allotted to the African lethargy which Nicolas described[4] in 1880 under the designation "Somnosis," a word now disused, which, if worth reviving, might fitly be applied to the similar constant somnolence of different nature.[5]

Most examples of so-called "narcolepsy" described by recent writers have occurred in hysterical subjects, sometimes after hysteroid convulsion, sometimes independently. Among the latter are cases in which the sleep attacks endured for hours, days, or weeks, and this condition was apparently such as in England has been termed "trance." To this, and also to post-convulsive sleep, the term is certainly inapplicable.

This brings before us another source of confusion, the

[1] Gelineau, *Gaz. des Hôp.*, 1880, Nos. 79 and 80.

[2] Foot, Acad. Med., Ireland, *Lancet*, i., 1887, p. 25.

[3] Caton, *Clin. Soc.*, Feb. 8, 1889.

[4] Nicolas, *Comptes Rend. Acad. de Science*, May 10, 1880.

[5] Gelineau speaks also of a "maladie de sommeil," which is the "sleeping-dropsy of the English." It would be interesting to know to what he refers.

danger of mistaking attacks of minor epilepsy for true sleep attacks. A patient with minor epilepsy, and sometimes with attacks that are more severe, who is aware of the commencing loss of consciousness, often describes his sensation as that of "going to sleep." But this is no evidence of the nature of the attack. It is common in unquestionable epilepsy. A recent writer on narcolepsy, Friedmann (of whose article an abstract appeared in this Review, 1906, p. 575), described fifteen personally observed cases. From my own experience, I should say that, on an average, before fifteen cases of narcolepsy were met with, 30,000 cases of epilepsy would come under observation. The malady may be more frequent in Germany, but a suspicion is raised that most of the cases were really minor epilepsy. This is supported by the statement that the usual duration of the attack was fifteen to twenty seconds, and that consciousness was often not completely lost.

It is wise to confine the use of a name to that to which it was first applied. The case I have narrated seems a typical example of the condition which "narcolepsy" was first used to designate and to which it should be restricted.

LANDRY'S PARALYSIS

By A. LEWIN SHEPPARD, M.B., B.S.,
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and
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THE rarity of cases which answer to the clinical picture originally described by Landry, and the association of a distinct micro-organism therewith, suggested our placing on record the following case, which was admitted to the Bristol Royal Infirmary, under Dr Shaw, to whom we are deeply indebted for permitting publication.

The patient, a lad of 17, was employed as a packer in some oil and colour works. He handled many tins, but no lead.

Previous history.—He was a teetotaller with no venereal taint, but he had never been strong. Three years ago he

had a diseased hip, and three months prior to admission he had quinsy. His doctor, who lanced it, states that there was nothing to suggest diphtheria, and a swab taken from the throat after admission showed only a slight growth of cocci.

Family history.—The father suffered from bronchitis and the mother was not strong. There were twelve children, of whom six were dead—four of pulmonary tuberculosis and two in infancy. The remaining five were healthy.

History of the present illness.—Seven days before admission he noticed slight numbness in the fingers, followed by similar sensations in the toes the following morning. Four days ago the numbness became more general, and there was some pain in the back, chest, and head; the legs becoming powerless, and walking impossible, while the arms were similarly affected. Since that time the loss of power had become more decided, and there had been increasing dyspnœa and dysphagia. According to the mother, two days ago, and the day before admission, the patient complained that he could not feel a hot-water bottle through blankets; but when, however, it was applied very hot, and directly to the feet, he said it went through him like needles. The rectum and bladder had been emptied normally.

Condition on admission.—The patient was of fair complexion and rather thin. He was quite intelligent. Temperature 99° F., pulse 72, and respirations 24. He was restless and cyanosed.

Mouth.—The tongue was dirty and the teeth were good, with no blue line on the gums.

Chest.—The heart and lungs were normal. Respiration was entirely diaphragmatic, with only slight movement of the false ribs. He was consequently unable to cough, or expel mucus or food from the mouth or throat.

Abdomen.—Soft and moving freely. Neither spleen, liver, nor enlarged glands felt. Marked aortic pulsation.

Muscular system.—Movements of the face and neck were normal, with the exception of the soft palate and the constrictors of the pharynx, both of which were almost completely paralysed. Phonation was fairly good. Both limbs were extremely weak, and both sides equally so. Movements at the elbow were stronger than those at the wrist and shoulder, but the arm could not be flexed or extended against the least pressure. He was

unable to raise his feet from the bed, but with the thigh supported he could extend the leg slightly. The muscles, therefore, of both limbs were stronger in the upper part. The intercostals, as mentioned above, were paralysed.

Nervous system.—Sight and hearing were both good, but the patient unfortunately died before the fundus had been examined. The other cranial nerves were normal. The pupils were equal and reacted normally. Sensation to touch, pain, heat, and cold were everywhere normal, and equal on the two sides.

Reflexes.—The knee-jerks were gone. There was no plantar reflex or ankle clonus.

The electrical reactions were normal to both the continuous and interrupted currents.

The urine was of normal quantity and acid, with a specific gravity of 1025. It contained no albumen or sugar.

The cerebro-spinal fluid obtained by lumbar puncture was clear; and under great pressure, after a syringe holding 10 c.c. had been filled and removed, another 23 c.c. ran out through the needle in a strong stream.

Of the few cells present, polynuclears were in excess; there were no endothelial cells, and very few lymphocytes. Some of the leucocytes contained Gram-positive cocci.

Culture in peptone broth yielded a Gram-positive coccus, occurring in very long chains. The following results were obtained on further cultivation. Slight clotting appeared in milk after forty-eight hours. Solutions of saccharose, lactose, maltose, glucose, and galactose were acidified; those of raffinose, salicin, and mannite were not altered. Slight acidity was present in inulin solution. A good growth appeared on gelatine at 20° C. Nitrates were not reduced. On solid media, such as agar-agar, glycerine-agar, nasgar, Conradi-Drigalski, etc., the organism retained its streptococcal characters, and the strain is still alive.

The blood-count gave 7,400,000 red cells, and 10,080 whites, with 98 per cent. of hæmoglobin, giving an index of 0·66. A differential count showed 70·00 per cent. of polymorpho-nuclears, 7·40 per cent. of large mono-nuclears, 22·20 per cent. of lymphocytes, and 0·40 per cent. of eosinophile cells.

Trophic changes.—None.

Progress and treatment.—The patient died eighteen hours

after admission, on the eighth day of the disease. He had strychnine hypodermically, and oxygen, which certainly relieved the dyspnœa, and nasal feeding was resorted to as deglutition became too embarrassed. He was quite rational up to the time of death, which took place quietly after a nasal tube had been passed. The heart failed with the respiration, but one or two voluntary breaths were taken afterwards with artificial respiration.

Post-mortem examination.—The lungs were œdematous, and there were a few old pleural adhesions on the right side. The liver and kidneys were intensely congested. The heart was normal throughout. There were no other features of note except the marked enlargement of the spleen, which weighed 14 ounces, and was very congested and extremely soft, and a similar enlargement of the mesenteric glands. There was no evidence of tuberculosis.

The spinal cord was congested throughout, the vascular change being most marked in the cervico-dorsal area. The cerebral meninges were also congested, but there was no exudate, and the brain tissue presented normal appearances.

Cultures made from the heart-blood yielded a variety of organisms, some of which resembled the streptococcus of the lumbar fluid, but, as the autopsy could not be performed until eleven hours after death, the results were discarded.

Microscopically, all the tissues were carefully examined for micro-organisms, but neither in the enlarged spleen, nor in the cord, etc., could any be detected. It is possible that this absence of the organisms might be due to their solution by the tissue fluids. The liver, lung, and kidneys showed active hyperæmia, and the early cloudy swelling.

In the cord, all the capillaries were widely dilated, and numerous extra-capillary hæmorrhages were visible. The cornual cells showed the various stages of chromatolysis and vacuolation. There was no degeneration of nerve fibres. The nerve sheaths were intensely congested, but no change could be made out in the nerve fibres by Donaggio's and Marchi's methods.

Remarks.—Since Landry's original account of the disease in 1859, many cases have been reported, but of these a large number may be excluded, as they differ in many cardinal points from the Acute Ascending Paralysis as then described.

Let us look for a moment at a summary of Landry's original description:

The patient, a man, during the past year had three or four attacks of fever, commencing sometimes with rigors. These were called "Fluxions of the chest," and were possibly pneumonic, with some pleurisy, as old pleural adhesions were found at the autopsy, but no tuberculosis. A fortnight after the last attack, from which he had hardly recovered, tingling sensations came on in the hands and feet; two weeks later a gradual ascending paralysis commenced, and a fortnight after this he died with respiratory failure. The chief points to be noticed were: an ascending progressive paralysis with only a slight rise of temperature, loss of reflexes, absence of sphincter trouble, no trophic or sensory changes, and no change in the electrical reactions. Death occurred from respiratory failure.

Many cases called Landry's paralysis have had pyrexia, or sphincter involvement, while others have shown sensory disturbances, or abnormal electrical reactions.

It is therefore obvious that it has never been very definitely laid down as to what exactly constitutes Landry's disease. Judson Bury,[1] in Allbutt's "System of Medicine," says: "I believe the clearest conception of the disease will be obtained by basing the description of the symptomatology on the historical case described by Landry in 1859; for surely if the name Landry's paralysis is to retain a place in the classification of nervous disease, it must be used to designate cases presenting the features described by the discoverer, and not in other senses adopted by some writers on the subject."

In the Goulstonian lectures of 1907, on "Toxic Conditions of the Nervous System," Farquhar Buzzard[2] described four closely similar conditions, viz. Acute anterior poliomyelitis, Acute ascending myelitis, Acute toxic polyneuritis, and Landry's paralysis. In the differential diagnosis he excludes Acute toxic polyneuritis by the presence of the sphincter and sensory disturbances; and in diagnosing Landry's paralysis from Acute anterior poliomyelitis and Acute ascending myelitis, he lays stress on the slight rise of temperature, the comparative absence of general symptoms, the slight pain in the back, with tingling in the extremities, the

[1] Clifford Allbutt's "System of Medicine," 1899, vol. vi. p. 695.

[2] *Lancet*, March 30, 1907.

unchanged electrical reactions, and the absence of trophic changes. As to motor phenomena, he contends that the paralysis is general and symmetrical, without wasting, while the trunk is affected, but the diaphragm escapes. He also makes a point of the extremely bad prognosis in Landry's disease. The condition itself he regards as possibly a species of spinal lymphatic intoxication.

In looking over the clinical features of the case we notice at all points a very marked similarity to Landry's original description; especially the coincident onset of symptoms in both upper and lower extremities; indeed, the tingling sensations are stated to have begun in the hands before the feet were affected. In all other respects the case agreed with what is now recognised as the classical description of Landry's acute ascending paralysis.

Certain points of interest were noticed. Firstly, the excessive pressure of the cerebro-spinal fluid; secondly, in the examination of the blood, the increased number of red and white cells, and the decrease in eosinophilous cells; thirdly, the presence of the organism in the cerebro-spinal fluid, its reaction with the various sugars, and its vitality. With regard to the "spinal lymphatic intoxication" in this case it may be noted that there was no evident primary focus for infection, either in the bladder, the extremities, or elsewhere. The presence of the streptococcus in the cerebro-spinal fluid, and its definite absence from the blood-stream, suggests the distribution of its toxin by lymphatic channels, a course which might explain its intense and comparatively localised action.

Abstracts

ANATOMY.

SOME QUESTIONS ON THE DEVELOPMENT OF THE CEREBRAL
(296) **CORTEX IN MAMMALS. (Alcuni questioni sullo sviluppo della corteccia cerebrale nei mammiferi.)** FRAGNITO, *Annali di Nevrologia*, Fasc. 5-6, 1906.

IN an earlier paper the author stated that the thin wall of the ventricle of the cerebral hemispheres was formed by numerous, more or less rounded, embryonal cells, but that at the tenth to

twelfth day this wall was divided into two layers, an internal and a thicker external, destined to form the cortex cerebri. A little later three layers could be distinguished, of which the internal and external were well provided with cells. The migration of cells from the internal or ependymal layer to the external layer is demonstrated by the presence of chains of cells, which extend uninterruptedly from the one to the other, across the intermediate layer, which contains very few cells; and also by the fact that although no karyokinetic figures are seen in the cortical layer, it steadily increases in thickness, while the ependymal layer, in which karyokinesis is most active, becomes thinner.

In the inner layer the cells have generally an elongated oval shape, and both ends are pointed. Towards the periphery of this layer some pyriform cells are seen, and these increase in number towards the outer layer, and their pointed extremity is directed towards the surface.

This apex of the pyriform neuroblast, according to His, represents the commencement of the axis-cylinder. From this view the author dissents. He has shown, in an earlier work, that the axis-cylinder is not an outgrowth from this apical process, and Paton and Hatai have demonstrated that the apex of the cell is directed towards the meninges. Moreover, the author has shown that the dendrites are developed before the axone, and also that the sparse protoplasm of the neuroblast, arranged around the nucleus, has no relation with the future prolongations, which are derived later from chains of cells.

It is more probable that the apex of these pyriform neuroblasts points in the direction in which the cells are migrating.

As in other parts of the central nervous axis, the single neuroblasts are not transformed into so many ganglion cells. Each ganglion cell is formed by the transformation of a more or less numerous colony of embryonal cells, and in the cortex of a dog, at the fiftieth day of development, many colonies are seen, which consist of one large clear nucleus, the primary neuroblast, and of a number of smaller cells, the secondary neuroblasts.

The author then discusses the question, whether all the secondary elements in each colony lose their individuality and become transformed into the protoplasm of the adult cell. This question is suggested by the existence of some corpuscles which are more or less intimately incorporated with the nerve cells or with their processes. Such corpuscles have been seen in several varieties of cells and in different animals.

To these cells Ramon y Cajal has given the name "Neuroglial Satellites," and the question arises: Have they anything in common with the secondary neuroblasts of the colonies described by the author?

The author does not consider that all the neuroglial elements spring from the ependymal cells, spongioblasts, but suggests that some of them are derived from the same elements which give rise to the nerve cells. He suggests, also, that a few of the secondary neuroblasts, instead of being transformed into the protoplasm of the nerve cell, may remain attached to the nerve cell, but undifferentiated. These he terms "Satellite Nuclei." Sometimes these satellite nuclei have been seen within the protoplasm of ganglion cells. For instance, in the lesions produced by tearing out the sciatic nerve in rabbits, the author and Donaggio found, in cells whose endocellular network was much altered, an accumulation of such nuclei, a kind of syncytium; and Modena has seen them in the cells of the cerebral cortex in a case of Huntingdon's chorea.

No cells resembling those included in the ganglion cells were present in the vicinity. It is difficult to see, therefore, how they could have penetrated from outside.

Further researches as to the nature of these cells are being carried on by the author. R. G. Rows.

PATHOLOGY.

CHOLINE IN CEREBRO-SPINAL FLUID. O. Rosenheim, *Journ.* (297) *of Physiol.*, Vol. xxxv., 1907, p. 465.

This paper is a further contribution to the question whether the presence of choline in the cerebro-spinal fluid and in the blood is a diagnostic sign of acute degenerative diseases of the nervous system. The cerebro-spinal fluid was examined for choline in fourteen cases of nervous diseases by means of micro-chemical methods. In four cases of general paralysis and in one case of hæmorrhage in the internal capsule, choline was found to be present. In two cases of meningitis and three cases of general paralysis no choline was found. It was also absent in two cases of dementia, one case of melancholia, and one case of senile dementia with herpes zoster.

The blood was examined in four other cases. Choline was not found in three cases of general paralysis; it was present in one case of organic dementia, accompanied by seizures.

From these results the author concludes that the presence of choline in the cerebro-spinal fluid and in the blood is a diagnostic mark of acute degenerative diseases of the nervous system. No details of the cases and of the post-mortem examinations are given.

The extracts of various organs which had been subjected to a special process of ether dialysis were examined for choline. This

base, which had been isolated before from brain extracts by Gulewitsch and Vincent and Cramer, was identified micro-chemically in brain extracts. It was also found in the extracts of the lung, liver, kidney, spleen and testis. It was not found in the heart and blood.

The author confirms the conclusions arrived at by Mansfeld, Vincent and Cramer, French and Allan, that the original micro-chemical method for choline recommended by Mott and Halliburton and Donath is fallacious. CRAMER.

THE FUNCTION OF THE PITUITARY BODY—EFFECTS OF
(298) **REMOVAL. (Recherches sur la Physiologie de l'hypophyse du cerveau. L'hypophysectomie et ses effets.)** N. G. PAULESCO, *Journ. de Phys. et de Path. gén.*, T. ix., No. 3, p. 441.

THE author has removed the pituitary body wholly or partially in a large number of dogs and cats. The method of operation is a new one. Access is obtained by the temporal route. The zygoma is resected, the temporal muscle incised. A large opening is made in the bone in the temporal region of one side, the dura incised and the brain raised and moved to the opposite side. This is facilitated by a second trephine opening upon the opposite side. The pituitary body is removed by a small curette. The experiments form three series: (1) total removal, (2) almost total removal, and (3) partial removal. In addition, control experiments were performed in which the parts were exposed and damaged to some extent without injury to the pituitary body.

Total removal of the whole gland is followed by rapid death, on an average in the case of dogs within twenty-four hours. Longer survival, occasionally occurring when the complete operation had been attempted, is to be explained by portions of greater or less size having been left behind at the time of operation. The test for the presence of sugar in the urine was positive in some cases, in others negative. In one experiment, where the gland was completely removed, it is noted that the dog did not pass urine before its death, which took place forty hours after operation. Partial operation is followed by results varying according to the part of the gland removed. Removal of the nervous part is compatible with prolonged survival and does not give rise to any appreciable symptoms.

The epithelial part of the gland is considered by the author to be divisible into a cortical portion and a medullary portion, the latter including the epithelium which is reflected upon the nervous part.

Removal of the whole of the cortical portion is rapidly fatal.

Its partial removal permits of continued survival without recognisable ill-effect. It is not possible to make sure of removing the medullary portion alone, but in one case of partial operation followed by survival for three months, the post-mortem examination revealed total absence of this part. Separation of the gland from the base of the brain is in its effects equivalent to total removal.

The author concludes in general that the pituitary body is an organ indispensable to life. Insufficiency of function as a result of total, or almost total, removal is not evidenced by any particular or characteristic symptom. Of the constituent parts of the gland, the cortical portion of the epithelial lobe is the most important functionally. W. A. JOLLY.

THE CHANGES IN THE PITUITARY AFTER EXTIRPATION
(299) **OF THE THYROID OR SUPRARENALS IN THE DOG. (Sur les modification de l'hypophyse après l'extirpation de la thyroïde ou des surrénales chez le chien.)** ALQUIER, *Journ. de Phys. et de Path. gén.*, Tome ix., No. 3, p. 492.

ROGOWITSCH observed hypertrophy of the pituitary in dogs and rabbits from which the thyroid had been removed. Hypertrophy has also been observed in the human subject in cases of myxœdema, cretinism, and exophthalmic goitre. Further, hypertrophy of this gland has been described by Boinet and others as following upon ablation of the suprarenals in guinea-pigs, rabbits, and cats. Various explanations have been offered, some attributing the hypertrophy to functional compensation, others to the effects of intoxication.

The object of the author has been to elucidate (1) whether the character of the hypertrophy is the same according as thyroid or suprarenals are removed, and (2) how, in the case of thyroidectomy, the date of its appearance is related to toxic phenomena, and especially to renal changes.

Twelve operations were performed in which thyroid and parathyroids were extirpated at one or at successive times, or in which a greater or less amount of thyroid or parathyroid tissue was allowed to remain. Hypertrophy of the pituitary resulted, ranging from a condition marked by increase in the number and volume of the acidophil cells, which are swelled by colloid, to a state of maximal alteration. Here, in addition to increase of acidophil cells, the colloid is much augmented. It is present as intracellular granules, and also as extracellular masses, irregular or rounded.

In five dogs the suprarenals were removed in two stages. Only very slight changes in the direction of hypertrophy were to be seen in the pituitary.

As a sequel to thyroidectomy, pituitary hypertrophy appears after the toxic changes in liver and kidney.

W. A. JOLLY.

CLINICAL NEUROLOGY.

LESIONS OF THE GANGLIA AND NERVE ROOTS IN HERPES ZOSTER.

(300) **(Les lesions radiculo-ganglionaires du Zona.)** DÉJERINE and ANDRÉ-THOMAS, *Revue Neurol.*, May 30, 1907.

IN this paper the authors deal with the conditions which are found in herpes zoster, as demonstrated by the method of Ramon y Cajal. In the case described, the herpes occurred in the region of the eighth dorsal nerve. In the corresponding ganglion and the central and peripheral nerve fibres, marked lesions of the nerve elements and of the interstitial tissue were found. The interstitial changes consisted chiefly of collections of lymphocytes, not in close connection with the vessels. These were more numerous in the peripheral pole of the ganglion.

In the nerve elements evidences of degeneration and regeneration were present.

In the central end of the posterior root most of the fibres had disappeared from some fasciculi, and those which remained were irregular in form and calibre. In others there were more fibres present, and the degeneration was less intense.

There was little infiltration or interstitial change; the meninges showed little sign of irritation.

In the ganglion most of the axis-cylinders were large, swollen irregularly, and moniliform; side by side with these were healthy fibres. All forms of irritative and degenerative lesions were seen.

In various regions, and especially near the capsule of the ganglion, numerous very fine fibres were found, which were twisted on themselves, and which ended in a swollen extremity. These sprang from the axis-cylinder between the cell and the seat of injury, and are compared by the authors to the arborised, regenerated fibres described by Ramon y Cajal in the cicatrices of cut nerves.

In the peripheral end of the posterior root and in the anterior roots similar appearances were found, but to a less extent. No micro-organisms were seen in the ganglion, but the lesions were

probably of infective origin. Some of the degenerations must have been due to the destruction of the nerve cells, but most of them must be attributed to a lesion which was initially parenchymatous.

R. G. Rows.

A CONSIDERATION OF SOME SYMPTOMS WHICH MAY BE (301) PRODUCED BY SEVENTH CERVICAL RIB. C. M. Hinds Howell, *Lancet*, June 22, 1907.

The author has collected 16 cases, which, in his opinion, illustrate the effects of accessory cervical ribs on the brachial plexus. All do not produce exactly the same symptoms, but 8 correspond very closely, and illustrate what may be called the typical symptom complex. (One must, however, remember that many cervical ribs give rise to no symptoms.)

Such symptoms are—

(1) Sensory, consisting in objective and subjective changes. The latter, almost invariably present, consist of pain from the shoulder to the hand, along the inner side of the arm, made worse by exertion or cold.

Objective changes consist in sensory loss, more pronounced for painful than for tactile stimuli, in an area corresponding to the first dorsal root distribution, but sometimes including the eighth cervical as well.

(2) Muscular changes. These in cases of long standing may be very marked.

There is slight loss of power in the extensors of wrist and fingers, more marked in flexors of wrist and fingers; of these the flexors of fingers suffer more than those of the wrist. The intrinsic hand muscles suffer most, the thenar eminence being as a rule more affected than anything else. Of the muscles here, the flexor brevis and abductor pollicis usually suffer most.

(3) Electrical reactions. If the case is at all recent there is typical R.D. in the affected muscles. This persists even in long-standing cases, provided the muscular atrophy does not progress too far, in which case all electrical response is lost.

(4) Cramps and spasms. These sometimes occur chiefly in the third and fourth fingers.

The diagnosis must be made with the aid of an X-ray photograph, but is not difficult if the condition be borne in mind.

Syringomyelia must be carefully excluded.

Other general points which the author indicates are—

(1) Sex. The majority of the patients are women. The explanation of this is difficult to see.

(2) Age. Ten of the sixteen cases occurred under the age of 30, *i.e.* during the period of most active growth and ossification. In those which occurred later, the possible ankylosis of the rib may account for the late production of symptoms.

(3) Other phenomena which have been attributed to cervical ribs—

(*a*) Stimulation of the sympathetic fibres in first dorsal root.

(*b*) Scoliosis.

(*c*) Affection of the upper roots of plexus (case reported by Déjerine).

The author finds that it is the short ribs, rather than the long ones, which cause symptoms, and also that though the ribs are almost always bilateral, the symptoms are more often unilateral. This he thinks entirely depends on the direction of growth of the ribs, those growing downwards and forwards being most likely to produce symptoms, probably by pressing the first dorsal root, between the first thoracic rib and their own lower border.

Treatment.—Two cases have recovered after operation, two have benefited without being cured.

AUTHOR'S ABSTRACT.

SUPERNUMERARY CERVICAL RIBS, AND THEIR EFFECTS ON (302) THE BRACHIAL PLEXUS AND SUBCLAVIAN ARTERY.

COLIN K. RUSSEL, *Medical Record*, Feb. 16, 1907.

THE author describes four cases, in three of which neuro-muscular symptoms existed. In the fourth, vascular changes were present alone. In one of the first three cases the symptoms were similar to those of writer's cramp, and it is quite possible that the extra rib played no part in their production. The patient was a male clerk, aged 35. The other two cases occurred in women, aged 27 and 24—the first a nurse, the second a sewing-maid. Both present certain features in common. Both patients complained of pain in the arms and cramps in their hands—symptoms worse in cold weather. In both the thenar muscles were wasted, the abductor pollicis suffering most; but the hypothenar muscles and interossei had not escaped entirely, and there was some weakness in the flexors of the fingers. In the first case there was a patch of complete anæsthesia, corresponding to the first dorsal root; in the second case there was no objective sensory change made out. The first case was operated on and a cervical rib removed, as a result of which the patient was at first worse, but four and a half months after operation had improved slightly so far as

sensation went, but the muscular wasting had remained the same.

The last case is a remarkable one. A woman, aged 31, had for nine years suffered from numbness and alternating cyanosis and pallor of the fingers of the right hand. This condition had increased during the last year. She had, a year previous to admission to hospital, placed her hand, "as a cure," in carbolic acid solution, 1 in 40; subsequent to this procedure the fingers swelled, and later became gangrenous.

On examination there was a definite mass made out on either side above the clavicles. Over this the subclavian pulsated. The skiagram showed what were taken for undeveloped first thoracic ribs. Owing to gangrene the right arm was amputated through the insertion of the deltoid, the brachial and radial vessels being found the seat of obliterative endarteritis, and blocked by an organised thrombus, which had become "canalised."

The patient subsequently committed suicide, and a dissection was made of the neck on either side. The subclavian artery on the right side distal to the thyroid axis was contracted and almost cord-like for two and a half inches. The author was unable to determine whether an accessory cervical rib or an abnormal first thoracic rib was present, but the eighth cervical root issued from between the first and second ribs present, of which, however, only twelve a side existed.

C. M. HINDS HOWELL.

(303) **OCULAR DISTURBANCES DUE TO PRESSURE UPON OR STRETCHING OF THE CERVICO-DORSAL SYMPATHETIC.** JOHN DUNN, *Archives of Ophthalmol.*, Vol. xxxvi., No. 3, p. 361.

The Spinal Eye.—In this condition the patient complains of inability to use the eye with comfort for near work. Correction of refractive errors gives little or no relief. Usually there is photophobia. The symptoms are bilateral, and the intra-ocular discomfort may be severe. On examination the anterior chamber is seen to be very shallow, there is a peculiar bowing of the iris, giving the appearance of a bulging lens, the pupils are contracted, but react to light and to mydriatics. The fundus is normal.

The author records three of his cases. In all he has found cervico-dorsal spinal curvature, except in one case, where, however, X-rays later demonstrated some spinal disease. The symptoms are apparently due to pressure on or stretching of the oculo-spinal sympathetic, and appropriate treatment gives relief, whereas treatment directly of the eyes gives little or none.

J. H. HARVEY PIRIE.

RECURRENT FACIAL PALSY, WITH REFERENCE TO CERTAIN
(304) **ETIOLOGICAL FACTORS.** T. J. ORBISON, *Am. Journ. of Med. Sci.*, June 1907, p. 892.

THE author's view is that in addition to the exciting cause of this common condition, usually exposure to draughts, there is always a defective digestive system and faulty kidney elimination, the lowered nervous tone being a secondary and consequent condition. Attention to the hygiene of the alimentary tract, and particularly reduction of chronic poisons, such as alcohol, tobacco, and excessive meat, is the most successful line of treatment.

J. H. HARVEY PIRIE.

SPONTANEOUS COMPENSATORY ANASTOMOSIS IN FACIAL
(305) **PALSY. (Contributo alla conoscenza delle anastomosi e delle supplenze funzionali dei nervi, a proposito di un caso di paralisi facciale periferica, curata col trapianto nervoso.)** P. BOSSI and A. IARDINI, *Rivista di patologia nervosa e mentale*, April 1907, p. 173.

THIS is a case of old-standing, right-sided facial palsy in a girl of 23, the result of suppuration in the middle ear at the age of 3½ years. Clinically there was total paralysis of mimic movements on the affected side, but on wrinkling the forehead, pursing the lips, etc., a few feeble movements were visible on the paralysed side in the frontalis, levator of lip and angle of nose and in the mental muscles. All the muscles capable of voluntary movement reacted feebly to faradism and to galvanism, without polar changes. Facio-accessory anastomosis was performed, the peripheral segment of the facial being sutured into a button-hole aperture in the sheath of the accessorius. At the operation, the facial nerve-trunk was found much attenuated, and electrical stimulation produced no contraction in the facial muscles. After the operation, in spite of the facial nerve having been divided, the muscles retained their power of slight voluntary movement as before. There must, therefore, have been some other path than that of the facial nerve through which voluntary impulses were transmitted.

This, according to the authors, seems to have been through the facial nerve of the healthy side. Corroborating this view they found, on stimulation of the healthy facial nerve, that not only the muscles of the sound side contracted but also certain muscles of the opposite side. This very remarkable case shows the possibility of compensatory anastomosis between nerve-trunks.

PURVES STEWART.

NASO-PHARYNX AS INFECTION CARRIER IN EPIDEMIC
(306) **CEREBRO-SPINAL MENINGITIS.** J. S. FRASER and JOHN D. COMRIE, *Scot. Med. and Surg. Journ.*, July 1907.

THE idea of carrying out this investigation into the epidemic in Leith was suggested by reading the work of Ludwig Jehle, an abstract of which appeared in the *Review of Neurology* (June 1907, p. 490). With regard to seaports such as Leith, if we substitute dock labourers for miners and ships below decks for coal-mines, it appears to be very probable that infection is spread as Jehle suggests, *i.e.* is carried home to the children by their fathers, who become infected at their work. The naso-pharynx was examined by Fraser and Comrie for the presence of the meningococcus in thirteen cases of the disease, with two positive results (15 per cent.), and in sixty-nine contacts with ten positive results (14 per cent.). Of these sixty-nine contacts, fifteen were fathers, and it is important to note that the meningococcus was found in five of these (33 per cent.), and in only five of the fifty-four other contacts (9 per cent.). Five of these fifteen fathers worked on one ship in dry dock, and the meningococcus was isolated from the air of the engine-room. Twenty-three contacts were examined with negative results. The method of identifying the organism was that recommended by Gordon.

Conclusions.—1. That hot, dusty, ill-ventilated atmospheres, which provide conditions favourable to the growth of the meningococcus and to the occurrence of naso-pharyngeal catarrh, are often associated with the dissemination of epidemic cerebro-spinal meningitis.

2. That the high comparative percentage of fathers whose naso-pharynx was found by us to contain the meningococcus, points to the fact that they probably are the carriers of the disease to their children.

3. That the chief incidence of the disease in the Leith epidemic was among children of the lower classes, and that this is in favour of a naso-pharyngeal infection.

4. That infection of the naso-pharynx is undoubtedly an important factor in spreading the disease.

5. That it is advisable to isolate all contacts and to carry out a bacteriological examination of the nose and naso-pharynx. If the meningococcus be present these cavities should be disinfected, and the contacts should only be allowed to leave quarantine after the naso-pharynx has been reported free from the meningococcus on two consecutive occasions. AUTHOR'S ABSTRACT.

LOCOMOTOR ATAXIA: A NEW THEORY AS TO ITS CAUSE.

(307) L. N. DENSLOW, *Med. Record*, June 15, 1907, Vol. lxxi., p. 985.

THE theory put forward by the author is based on his "clinical experience of a number of cases." It is, briefly—that the pathological changes that occur are due primarily to irritation of peripheral nerves producing reflex disturbances in the spinal cord and brain, and that such irritation kept up continuously for a sufficient length of time can and does produce pathological changes in the cord, not merely functional disorders. Predisposing conditions are necessary; this usually means syphilis, but a neurotic temperament, alcoholism, sexual excess, over-exertion, etc., may suffice. But some peripheral nerve irritation is always present in the author's cases, both male and female; it has always been genito-urinary, and if this is treated the symptoms (tabetic) are relieved or may be even cured.

This persistent and continuous peripheral irritation, he believes, produces a coagulation of the colloid fat in nerve fibres, and, given this, the subsequent alterations are but natural sequelæ.

It is to be hoped that the author will record his cases in detail, so that a better estimate of the value of the treatment, and of the etiology of tabes, may be formed. J. H. HARVEY PIRIE.

THE CO-EXISTENCE OF TERTIARY SYPHILITIC LESIONS AND TABES. (Sur la co-existence des accidents syphilitiques tertiaires avec le tabes.)

(308) DUBOIS, *Thèse de Lyon*, 1906.

THIS thesis, which should be compared with that of Dorleans (abstracted in *Review of Neurology*, 1906, p. 748), is based on the consideration of 81 cases in which characteristic lesions of the skull, palate, eye, skin, cellular tissue, and testes were associated with tabes. Tertiary lesions may appear at the onset of tabes or after the latter has been existing for some time. In a case reported by Fournier a tertiary lesion occurred 15 years after the onset of tabes. In several cases the primary or secondary lesions had passed unobserved and untreated. In all but three cases in which there was either a cure or an amelioration of the tabetic symptoms, and in particular a return of the knee-jerks, anti-syphilitic treatment had no action on the tabes, though it produced a complete cure of the tertiary lesions. J. D. ROLLESTON.

ERB'S SYPHILITIC SPINAL PARALYSIS. [Die syphilitische Spinal-
(309) **paralyse (Erb).]** WIMMER, *Deutsche Zeitschr. f. Nervenheilk.*, June 1907, Bd. xxxii., S. 308.

WIMMER comprehensively reviews the problem as to whether the defining this disease as a separate entity is justified. He reported three cases. In the one of these that came to autopsy the following findings were made: Diffuse combined sclerosis of practically all the cord tracts, focal lesions in the dorsal region that could account for only some of the above-mentioned degeneration, with slight changes in the meninges and vessels. This is the seventh such case with these findings.

He concludes that although the affection obviously belongs to the same group as cerebro-spinal syphilis, yet it can be differentiated, both clinically and anatomically, from the other members of this group. The distinctive clinical features are: incomplete paraplegia, with spastic gait; great exaggeration of all the reflexes (Babinski's sign, etc., being present as well), combined with hypotonia—a most important diagnostic feature; early bladder symptoms; sensory disturbances (slight general hypoæsthesia in the lower extremities, there being no dissociation and no root or segmental distribution); pain (a prominent symptom); no muscle atrophy; integrity of the arms, pupils, eyes, and all cranial nerves. The course is progressive but not rapidly so.

Post-mortem, only four cases have revealed a pure pyramidal degeneration, the above mentioned picture being the one usually seen. ERNEST JONES.

TWO CLINICAL OBSERVATIONS OF PSEUDO-BULBAR PARA-
(310) **LYSIS WITHOUT PARALYSIS OF THE LIMBS. (Deux observations cliniques de paralysie pseudo-bulbaire sans paralysie des membres.)** H. LAMY, *Rev. Neur.*, Feb. 28, 1907.

BOTH patients were arterio-sclerotic individuals over 60, and presented the same syndrome—sudden appearance of dysarthria, the speech resembling that in bulbar paralysis; weakness of the muscles supplied by the inferior division of the seventh nerve, the platysma not being affected; great contrast between the speech difficulty and the comparatively free movement of tongue, lips, soft palate, and larynx; no difficulty in swallowing. One patient had slight weakness of the right hand. Both presented in a marked degree the facile and explosive laughter of the pseudo-bulbar. C. MACFIE CAMPBELL.

PRIMARY CORTICAL PARENCHYMATOUS CEREBELLAR ATROPHY. (Atrophie primitive parenchymateuse du cervelet, à localisation corticale.) (311) ROSSI, *Nouv. Icon. de la Salpêtrière*, Jan.-Feb. 1907, p. 66.

THE case on which this anatomo-pathological study is based is one recorded by Crouzon in his thesis on combined sclerosis of the cord. A résumé of the clinical aspect of this case and of two others shown by Marie is given. The condition comes on after the age of 60 ; it is slowly progressive, and recognised especially by disturbance of gait—slowness, titubation, a cerebellar character, but with also slight, though distinctive, ataxia and spasticity. There is also some muscular asynergy and disturbance of speech. The knee-jerks are exaggerated, and the plantar reflex of the extensor type. In one of the cases there were sensory and sphincter disturbances.

The cord was found to show only very slight changes, chiefly rarefaction in the posterior columns. The medulla, pons, cerebrum, and meninges were normal. The cerebellum was markedly diminished in size; this diminution affected all parts, but not everywhere to the same degree. Where most marked, the molecular and granular layers were much diminished in thickness, and Purkinje's cells had practically disappeared. In other parts the atrophy was limited to the granular layer and the cell of Purkinje, while in others only the Purkinje cell showed atrophic changes. The lesion was entirely limited to the cortical grey matter. In this case the symptoms developed after an enteritis; in the others no apparent cause was found. J. H. HARVEY PIRIE.

TUMOUR OF THE CEREBELLUM IN A DOG, ASSOCIATED WITH FORCED MOVEMENTS. (312) F. E. BATTEN, *Brain*, 1906, p. 494.

THE interest and value of the case lie in the attitude assumed by the animal and in the forced movements—rotation on his long axis and circus movements.

Attitude.—When left alone, the dog lay on the ground on his right side, with the right side of the face against the ground. The whole body was curved, the concavity being ventralwards and on the left side of the body. The position of the head was very marked and constant: the right ear was approximated to the right shoulder, and the head was rotated on its long axis so that the left side of the face was turned up. The right fore-limb was abducted to an angle of about 45° to the long axis of the head, the left fore-limb was flexed, and the forepart lay parallel with the long

axis of the dog; the hind-limbs were generally flexed and rigid (seemingly due to volitional effort to avoid forced movement).

Forced movements.—If disturbed, the dog rotated on his long axis from left to right, *i.e.* as an outcoming screw (the head of the animal corresponding to the head of the screw), or, in other words, the right shoulder moved forwards across the trunk. Batten strongly objects to the term "rotation to the right or left" as ambiguous and misleading.

Circus movements.—The dog tended to move round in a circle on the floor anti-clockwise (the watch being supposed to be placed on the floor so that the observer can see the hands), the centre of the circle corresponding approximately to the hip-joints, and the radius extending from here to the tip of the nose. It was impossible to tell to what extent the movement was volitional.

There was no nystagmus or ocular paralysis. Knee-jerks were present and equal.

Pathological examination showed a larger sarcomatous tumour (or inflammatory lesion) involving the right lateral lobe of cerebellum and right side of medulla, and a smaller tumour on the left side of the cerebellum, affecting the cortex but not extending into the middle of the lobe.

The symptoms observed in this dog are compared with those recorded after experimental lesions on the cerebellum.

A. W. MACKINTOSH.

THE CEREBELLAR ATTITUDE AND THE DENTATE NUCLEI.
(313) E. FARQUHAR BUZZARD, *Brain*, 1906, p. 508.

BUZZARD records the case of a woman, aged about 60 years, suffering from carcinoma of the sigmoid end of the colon. She complained of general malaise and appeared depressed for a few days, and then became drowsy. Next day, when seen by Buzzard, she was quite unconscious. The attitude was markedly "cerebellar." "She lay flat on her back, with her legs rigidly extended and her arms rigidly flexed across her chest, the partially closed hands pointing upwards towards her chin. The neck was stiffly extended, so that in placing a hand behind the occiput and pressing forwards there was very little flexion of the head on the trunk, but a tendency to lift the shoulders as well as the head." The pupils reacted to light; the axes of the eyeballs were parallel in the mid-line, and the fundi were normal. Abdominal reflexes were absent; plantar reflexes were sometimes of the extensor type; the tendon reflexes were brisk. A pin-prick produced practically no response in any region. The pulse was small and of low tension.

Ponto-cerebellar thrombosis was diagnosed. The patient died the following day, and autopsy showed—apart from the condition of the cerebellum—no microscopical evidence either of arterial disease or of softening anywhere in the brain. In the cerebellum there was marked softening, due to recent thrombosis, limited to each dentate nucleus and its immediate environment. The cause of the thrombosis was evident, all the arterial branches supplying the dentate nuclei being the seat of disease, involving the intima but more especially the media (calcareous degeneration). Practically no normal cell was found in the dentate nuclei.

Buzzard thinks that the cause of death was not the lesion in the cerebellum, but toxæmia, which so frequently causes a fatal termination in cases of malignant growth. This toxæmia is associated with lowered blood-pressure, which would predispose to thrombosis in degenerated vessels such as were present in this case in the vessels supplying the dentate nuclei. As a general rule, no particular attitude is assumed or maintained by patients who become unconscious from the toxæmia associated with new growths. Buzzard therefore thinks that the lesion of the dentate nuclei and the attitude of body assumed by his case during the last thirty-six hours of life were in some way associated. The lesion must have caused a loss of function, not an irritative effect, and the resulting attitude may well be regarded—according to Jackson's hypothesis—as due to the more or less completely unopposed action of the cerebral middle level centres upon the lower centres in pons, medulla and cord, the cerebellar influence being largely cut off by the lesion.

A. W. MACKINTOSH.

CASES OF SPONTANEOUS INTRACRANIAL HÆMORRHAGE
(314) **ASSOCIATED WITH TRIGEMINAL NÆVI.** HARVEY CUSHING, *Journ. of the Am. Med. Assoc.*, Vol. xlvii., pp. 178-183.

CUSHING records three cases in which nævi occurring on the face followed closely the distribution of one or more branches of the fifth nerve, and were in two cases certainly, and in the third probably, associated with a nævoid condition of the dura mater. This condition of the dura had led to intracranial hæmorrhage followed by epilepsy in each case, with contralateral spastic paralysis in two cases. In two cases operation was done, with marked benefit in one case in which adhesions were separated.

Cushing states his opinion that such nævi are due in all probability to some congenital defect or intra-uterine lesion which has involved the Gasserian ganglion or its sensory root, and inci-

dentally states his belief that most if not all of the cases of spastic hemiplegia or paraplegia dating from birth are due to intracranial hæmorrhage, often cortical, and not to an inflammatory process.

He formulates his conclusions as follows:—

Vascular nævi occurring on the face show a tendency to conform in outline with the distribution of one or more of the main trigeminal branches.

These cutaneous nævi may be associated with some degree of hypertrophy of the deeper tissues of the face, with an enlargement of the eye, and finally with the presence of a corresponding nævoid condition of the dura mater. This dural nævus may lead to a spontaneous hæmorrhage during infancy, with consequences similar to those which follow the subdural hæmorrhages occurring during the early months of life from other causes—epilepsy, spastic hemiplegia, or even amentia.

Absorption of the clot may lead to cortico-dural adhesions, which in favourable cases can be separated with benefit to the epileptiform convulsions, provided measures such as carotid ligation are taken to prevent complications from too great a loss of blood during the operation. J. W. STRUTHERS.

PERIPHERAL OBLITERATING ARTERITIS AS A CAUSE OF
(315) **TRIPLEGIA FOLLOWING HEMIPLEGIA.** C. W. BURR, *Journ. Nerv. and Ment. Dis.*, Jan. 1907.

OBLITERATING arteritis is a not very infrequent, but much neglected cause of paralysis, which, when occurring in a case already affected by hemiplegia, may give rise to a triplegia apt to be erroneously attributed to some additional cerebral or spinal lesion.

The present case is that of a man, aged eighty years, who shortly after the onset of a right hemiplegia, with aphasia, became completely bedridden, the right arm and both legs being in a state of marked contracture. This condition slowly increased up to the time of his death, which occurred some months later. Examination of the brain showed sclerosis of the island of Reil, and an area of degeneration in the posterior limb of the internal capsule on the left side, with secondary degeneration of the pyramidal tract, but no lesion on the opposite side. There was marked irregular thickening of the intima, and sclerosis, with calcification of the media in both external iliac arteries. The walls of the popliteal arteries were also greatly thickened, and they were filled with recent thrombus. In cases of this kind, where there has been no preceding hemiplegia, the diagnosis is easy; but when, as in the present instance, the two are combined, the result somewhat resembles a severe spastic myelitis. HENRY J. DUNBAR.

ON SLEEP. (**Über den Schlaf.**) PAUL KRONTHAL, *Neurolog.* (316) *Centralbl.*, June 16, S. 553.

AFTER a tedious account of the more obvious phenomena of sleep, Kronthal expresses his conviction that the condition has nothing whatever to do with the nervous system. He bases this mainly on the opinion that sleep occurs in animals with no nervous system, and in plants. He divides the causes of sleep into three: one physiological—fatigue, and two pathological—poisons and cold. ERNEST JONES.

ETYMOLOGY OF THE EXPRESSIONS "CRAMPUS" AND (317) **"KRAMPF."** (**Zur Etymologie der Ausdrücke: "Crampus" und "Krampf."**) P. NÄCKE, *Neurolog. Centralbl.*, June 16, S. 546.

NÄCKE, who has done so much work on local cramp, here seeks to establish the origin of the above two words. He concludes they both arise from an ancient Aryan root, though there is no indication of them in Sanscrit. "Crampus" is not found in classical Latin, and the middle Latin word is probably derived from an older Teutonic word. Originally the terms indicated something bent or drawn together; only later were they applied in their present sense. Both terms were at first used in a more general sense, and only later has crampus been restricted, as in the sense of the English word cramp. ERNEST JONES.

PARAMYOCLONUS MULTIPLEX AS A COMPLICATION OF (318) **TYPHOID FEVER.** (**Di una rara complicazione della febre tifoide.**) GIACOMUCCI, *La Clin. Med. Ital.*, March 1907, p. 174.

Two brothers, whose family history was negative, were attacked with typhoid fever. In the youngest, aged 4, the disease was of a moderate intensity, and there was only a faint indication of paramyoclonus. On the tenth day convulsions occurred, followed by aphasia. On the twelfth, there was slight spasm of the masseters and pharyngeal muscles, rendering deglutition difficult. On the fourteenth, there were rapid antero-posterior and lateral movements of the head, which only stopped in sleep, and diminished in intensity when the patient was told to keep still. On the twenty-third, the temperature became normal, and the movements of the head almost entirely ceased. Speech

returned gradually. It was not till the thirty-fifth day that the child completely recovered this faculty.

The other boy, aged 14, became very restless on the eighth day of his disease. On the tenth, aphasia developed. On the twelfth, trismus supervened, with incontinence of urine and fæces, and spasm of the flexors of the right knee. On the sixteenth, there were rapid lateral and antero-posterior movements of the head. The next day the muscular contractions affected all the voluntary muscles, those of the face alone excepted. The contractions were clonic, very rapid, inco-ordinate, and arythmical, ceased at longer or shorter intervals, disappeared entirely during sleep, diminished when the patient was told to keep still, and increased whenever he saw the doctor enter the room. The fever came to an end about the thirty-fifth day, but the muscular contractions continued for some time longer, complete control over the muscles being not regained till the sixtieth day. Ptosis, which had developed simultaneously with the paramyoclonus, slowly disappeared. Recovery of language was also very gradual. During the first few days of convalescence, the boy could not read the word "mamma," although he could pronounce it, and was unable to write more than two syllables of his name (Sebastiano). Thus aphasia was associated with word blindness and agraphia. The aphasia and the paramyoclonus, which were of simultaneous development, are assigned a common cause, viz., a perturbation of the nerve-cells of the cortex compatible with their further existence and restoration of equilibrium.

J. D. ROLLESTON.

CUTANEOUS REFLEXES IN THEIR RELATION TO TENDON
(319) **REFLEXES IN TABES. (Les réflexes cutanés dans leur rapports avec les réflexes tendineux au cours du tabes.)** NOICA and STROMINGER, *Presse Médicale*, April 27, 1907, p. 266.

THE writers examined eighteen tabetic patients whom they placed in two categories. The first contained six patients in whom there was a very advanced degree of ataxia. In this class the knee and ankle jerks were completely abolished, and the cutaneous reflexes were almost completely lost. In the second class were placed twelve patients in whom the disease was less advanced. In all, the knee and ankle jerks were abolished, except in one, in whom hemiplegia had supervened, and who still preserved his knee jerks. In six all the cutaneous reflexes were present; in the other six some of the cutaneous reflexes were abolished; but in each case only the lowest reflexes, viz., the

plantar on one or both sides, the gluteal on both sides, the anal, and in one case the lowest division of the abdominal reflex on both sides. The writers conclude that the state of the cutaneous reflexes is not related to the age of the individual or to the duration of the disease, but to the degree of intensity of the affection, and that the lower reflexes are affected long before the upper. J. D. ROLLESTON.

THE TENDON-REFLEXES OF STRAINED MEMBERS. (Die Sehnenreflexe angestrengter Körperteile. Untersuchungen an Marathonläufern.) (320) MILT. OECONOMAKIS, *Neurolog. Centralbl.*, June 1 and 16, pp. 498 and 563.

THIS article is based on Edinger's well-known Ersatz-Theorie, and contains an account of the effect of forced exercise on certain reflexes. Forty-five of the competitors in the Marathon race were examined, forty-three before the race and two only after it. The state of the knee-jerk, Achilles-jerk, pupil reaction to light, and the size of the pupil were noted. The jerks were found increased in six individuals, the knee-jerks alone in two more. The knee-jerk was absent on both sides in one individual, diminished on both sides in three, and on one side in one. The Achilles-jerk was diminished on both sides in three, on one side in four. This contrasts with Knapp and Thomas' examination of forty-nine runners, in whom increased reflexes were present in forty-four.

Fourteen individuals were tested both before and after the race. Before the race the jerks were normal in nine, active in one, diminished in four; after the race the knee-jerks were increased in six, diminished in four, and lost in four; while the Achilles-jerks were increased in eight, diminished in five, and lost in one.

No pupillary abnormalities were observed in any individual.

It is noteworthy that the diminution and loss of the jerks were found almost exclusively in the Greek competitors. This the writer attributes to the fact that they were untrained peasants in whom the strain was therefore more manifest; they also used alcohol, while the foreigners did not. Some general considerations on fatigue conclude the article. ERNEST JONES.

CONCERNING THE MIRACLES OF LOURDES. (A propos des miraculées de Lourdes.) (321) J. ROUX, *La Loire Médicale*, June 15, 1907.

A STATEMENT by the writer that he had had two patients cured at Lourdes, and that he sincerely pitied them, called forth a letter couched in ironical terms from a correspondent to *The Tribune*.

The present paper gives reasons for the pity expressed, and discusses the question of the Lourdes miracles in general. One patient, a servant girl suffering from paraplegia, was cured after her fourth immersion at Lourdes, but shortly after her return developed a violent rabiform hysteria. The "miracles" constitute simply one form of suggestive treatment, and as such will be condemned by almost all neurologists. Apparent cure by any form of suggestion is almost always followed by a reappearance of the disease in a different guise, and the relapse is generally more serious than the original attack. Even as a means of suggestion, the author, who has been to Lourdes, cannot find terms strong enough to express his opinion as to the "miracles," carried out as they are in a manner calculated to trade on the ignorance and superstition of the "pilgrims" for the grossest and most obvious commercial purposes. HENRY J. DUNBAR

PSYCHIATRY.

PERIODIC PSYCHOSES AND EPILEPSY. [**Psychoses périodiques** (322) **et Épilepsie (considerations cliniques et anatomo-pathogiques**).] ANGLADE and JACQUIN, *L'Encéphale*, June 1907.

THE authors find that etiologically the dominating predisposing cause in both cases is heredity; an exciting cause is often difficult to assign, but those which are operative in the one class are equally so in the other. They enumerate emotional states, modifications of climate, nutritional disturbances, infections, intoxications and disorders of genital functions, assigning to menstruation an important rôle.

Clinically there is a strong resemblance between the character and conduct of the subjects in the two classes, not only between, but during the access of attacks. The return of the access in periodic mania may be ushered in by signs, which, like the aura in epilepsy, although varying in different cases, are always identical in the same individual, *e.g.*, intercostal pain, visual hallucinations, persistent hiccough, or peculiar gestures or acts. The successive attacks in the periodical psychoses also, as in epilepsy, resemble one another in the same individual. Although there is never found the loss of memory which is so characteristic after epileptic crises, they remark that in the epileptic psychoses which are most closely analogous to intermittent psychoses, this symptom may be slight or absent.

Various cerebral congestive signs have been described in periodical psychoses, such as redness, swelling and heat of face, vertigos, short loss of consciousness, slight convulsive movements,

momentary embarrassment of speech, incomplete partial paralyses, transitory hemiplegias—symptoms which, in conjunction with the etiological factors mentioned, appear to them to justify the statement that between epilepsy and periodical psychoses there is a close bond of connection. They do not assert that *all* periodical manias are of epileptic nature, but that in many cases the striking analogies can only be explained by identical processes, the localisation of which alone differs. Their anatomo-pathological findings strengthen still more this view. They contrast the form of epilepsy in which occurs diffuse cerebro-sclerosis with periodical insanity. In these latter cases they find macroscopically resemblances to epileptic brains, especially as regards zones of induration, either superficial or deeply situated; microscopically they have constantly found neuroglial proliferation. They draw special attention to the close resemblance between the subcortical neuroglia in the brain of a two-year-old child and that in periodic insanity, and they believe that the adult brain attacked by periodic insanity or epilepsy in so far as concerns the neuroglia is in a state of evolutive activity, which will be exaggerated under adverse influences, infections, toxines, etc.

The important fact in the two diseases is evidently the special constitution of the subject, attributable to some anomaly of development, which they believe to be the infantile character of the neuroglia. They suggest that there is a disturbance of equilibrium between the vitality of the nerve-cell and that of the neuroglia, so that in proportion to the activity of the latter is the feebleness of the former. Hence the morbid agencies which cause the mental decay respect the neuroglia or augment its activity. Possibly paralytics or seniles may transmit to their descendants a neuroglia of a particular susceptibility which may constitute the pathological anatomy of the predisposition. According to this hypothesis the convulsive aptitude of Joffroy, and the predisposition to periodical psychoses will be nothing else than a defect of equilibrium between the vitality of two tissues of the central nervous system, and if the neuroglia gains the upper hand the result will be epilepsy or insanity. JOHN TURNER.

A CASE OF LOSS OF MEMORY. C. W. BURR, *Amer. Journ. of* (323) *Insanity*, Jan. 1907.

CASES of loss of memory, unaccompanied by other signs of dementia or evidences of physical disease, are somewhat rare. The case here recorded is that of an iron-moulder, aged about 55 years, who was admitted to the Philadelphia General Hospital six years ago, having been found wandering about the streets in a

confused state and unable to give any account of himself except his name and trade. After admission he had an acute violent attack lasting for a few days, but since then his condition has been normal, with the exception that he has complete loss of memory of many years of his life and total inability to remember anything happening at the present time for even a few minutes. He recollects well many of the incidents of his early life, and knows that he has been married, but has no recollection of his wife. He writes well, but forgets the end of a long sentence while he is engaged with the first clause. He reads aloud correctly, but has no recollection of the beginning of a paragraph by the time he has got to the end of it. He converses intelligently. His conduct is irreproachable. He has never learned the way to his own bed, and does not know where he is, what day or year it is, or whether he has recently partaken of food or not. The writer discusses the question of the patient's insanity in the technical sense, and the extent of his responsibility for his actions. If he committed murder he would know that he was doing wrong, but would forget the incident in a few minutes, and, if condemned, would not know the reason for his punishment. He could not make a will, as he does not know whether he has any property or any relatives, and if told about them would forget the first things while the later ones were being explained.

HENRY J. DUNBAR.

CLINICAL OBSERVATIONS OF AMBULATORY AUTOMATISM.

(324) **(Automatisme ambulatoire; observations cliniques.)** P. COURTON, *Ann. méd.-psych.*, Jan.-Feb. 1907.

FIVE cases of ambulatory automatism in various disorders are reported. The first patient, a hysterical man of 41, for sixteen years had had periodic attacks of an uncontrollable impulse to wander; in contrast with the typical hysterical episodes of this nature there was no subsequent amnesia. The wandering episodes replaced as "equivalents" the periodic attacks of malarial fever to which the patient had been subject. During the period of the wandering he acted with a certain amount of intelligent deliberation.

The second case was a hystero-epileptic of 22, in whom the episodes took the form of nocturnal somnambulism with complete amnesia for the period of wandering.

The flight of the third patient, an alcoholic harassed by auditory hallucinations, has nothing in common with the two previous cases. The fourth case was one of dementia præcox; the wandering episodes here were as little explicable as the majority of the

bizarre, incongruous reactions in dementia præcox, and are placed by the author under the motor disorders of this disease. Perhaps a more thorough analysis would enable one to see in these cases that the wandering is essentially a distorted reaction to certain mental contents, and should be grouped symptomatologically along with the preceding case.

The fifth case was that of a defective, whose desertions from military service were merely the expression of constitutional instability, and not of a deteriorating psychosis; when placed in a suitable environment the patient was able to adjust himself satisfactorily. C. MACFIE CAMPBELL.

AMBULATORY AUTOMATISM. HUGH T. PATRICK, *Journ. Nerv.* (325) *and Ment. Dis.*, June 1907.

THIS is an exceedingly interesting article, read by the author as President's Address before the American Neurological Association at Washington last May. In his introductory remarks he alluded to this syndrome as following trauma, epilepsy, in alcoholism, and also as occurring in hysteria and as an epileptic equivalent. He was, however, of the opinion that some cases could not be included in any of these categories, but appear to be striking manifestations of psychic instability. He gave notes of seven cases which had been under his personal observation in support of his views. None of these cases were, in the author's opinion, epileptic in origin, and in referring to a very extensive casuistic from many authors he took occasion to question the diagnosis in very many reported cases of epileptic flight, although he does not dispute its occurrence. In all of his cases the subjects were of originally unstable nervous constitution, and prior to the flight were worried or in financial or domestic straits. In each case the flight was in harmony either with an unsettled vagabond disposition or the exigencies of the moment. The flights were of varied duration, and followed by complete or partial amnesia.

In three cases under hypnosis a true account of the subject's wanderings was obtained. Four were distinctly hysterical in origin, but the others fitted into no definite category. In reviewing the whole literature, Dr Patrick was struck by the enormous preponderance of men, whereas the incidence of epilepsy is about equally divided between the sexes; also bromide in no case gave relief; and further, the frequency with which automatic fugues have as exciting factor casual conditions—debt, domestic quarrels, threatened arrest, etc.—is, in the author's opinion, distinctly against their epileptic origin. R. CUNYNGHAM BROWN.

ATTACKS OF EPILEPTIC PETIT MAL WITH PARAMNESIC
(326) **AURA; ILLUSION OF FALSE RECOGNITION. (Crises de petit mal épileptique avec aura paramnésique; illusion de fausse reconnaissance.)** J. SÉGLAS (of Bicêtre), *Rev. Neurol.*, Jan. 15, 1907.

THE patient, twenty-eight years of age, had for four years been subject to peculiar episodes; without warning he would suddenly have the impression that what was passing around him was the duplicate of some scene previously experienced. Occasionally it formed part of a hypnogogic dream, and if he sat up for a few seconds the feeling disappeared and he could immediately go to sleep again. Violent emotional shocks and fatigue were apt to bring on such episodes. Not only did these attacks occur in the above uncomplicated form; in addition they occasionally formed the first stage of a typical attack of petit mal. The pseudo-recognition was not accompanied by the distress which is observed frequently in similar cases.

Other forms of attack to which the patient was subject made the diagnosis of epileptic petit mal clear, and the pseudo-recognition in some of the attacks evidently played the part of aura.

C. MACFIE CAMPBELL.

ACUTE TRANSITORY MENTAL DISORDERS ON THE BASIS
(327) **OF CEPHALALGIA AND HEMICRANIA. (Disturbi psichici acuti transitori su base cefalalgica ed hemicranica.)** M. A. BIOGLIO (of Rome), *Riv. di Pat. nerv. e ment.*, Jan. 1907.

THE observations of three cases in which at the acme of severe recurrent headache or hemicrania hallucinations of varying complexity were present. In all the three cases there was a bad family history. In the first case the presence of an intermittent delirium with complete amnesia for the delirious period suggested the existence of epilepsy. In contrast with these cases the author reports briefly the observation of a case of psychical epilepsy with severe headache as an aura. C. MACFIE CAMPBELL.

THE HÆMO-LEUCOCYTIC FORMULA IN THE ACUTE CON-
(328) **FUSIONAL PSYCHOSES. (La formula emo-leucocitaria nelle psicosi acute confusionali.)** O. SANDRI (of Florence), *Riv. di Pat. nerv. e ment.*, Feb. 1907.

THE acute confusional psychoses present a close affinity with toxic psychoses both in their symptomatology and in the pathological

changes in the cortex; usually some definite cause can be elicited, although the immediate cause of the mental disorder and of the accompanying somatic symptoms is not clear.

Some chronic psychoses, such as dementia præcox, may begin with a condition similar to the acute confusional psychoses of supposed toxic origin.

The author has investigated the cellular elements in the blood both in states of mental confusion, forming an episode on a chronic mental disorder, and also in states of primary mental confusion running quite a different course.

In this communication he gives the latter results, viz., the results of the examination of the blood in states of primary mental confusion in relation to tuberculosis, furunculosis, articular rheumatism, suppurating wound, pregnancy, lactation, gastro-enteritis, etc., and also where no somatic cause was elicited. He found that the alteration in the cellular composition of the blood was similar in all these cases, and concludes that there must be one pathogenic factor; this latter is probably not the direct product of the various toxic-infective agents, but probably has developed on the ground prepared by these latter agents.

This general result is based on the following observations, verified on a material of thirty cases:—

1. In the beginning of a case of primary mental confusion, the red blood corpuscles increase, the hæmoglobin rises, and there is a marked leucocytosis. The neutrophile polymorphonuclears increase, the eosinophiles are decreased or disappear, the large mononuclears become very scanty. The changes in the blood bear a direct relation to the severity of the case.

2. In the most severe cases the blood may retain the above cellular relations for months; in favourable cases it tends quickly to return to its normal composition.

3. The return of the blood to its normal cellular composition goes on steadily in favourable cases, even when the mental condition shows no sign of improving. On reaching its normal constitution, the blood does not keep the equilibrium, but there is a subsequent fall of the number of the reds and of the hæmoglobin, the polymorphonuclears diminish, while there is an important mononucleosis. As a rule, during this period the mental condition of the patient improves.

4. The blood may not return to its normal constitution until some time after the complete disappearance of mental symptoms. The author considers that acute confusional psychoses depend upon a certain constitutional peculiarity of reaction, and on a condition of intoxication. The severity of the condition does not depend so much on the primary intoxication as on the secondary toxins elaborated by the usual pyogenic inhabitants

of the intestines. The nature of the cellular changes points to such a pyogenic infection. C. MACFIE CAMPBELL.

PAROXYSMAL OCCURRENCE OF FEVER AND POLYNUCLEAR
(329) **LEUCOCYTOSIS IN GENERAL PARALYSIS. (Ueber paroxysmale Fieberzustände bei progressiver Paralyse mit Vermehrung der polynukleären Leukozyten im Bluté und in der Zerebro-spinalflüssigkeit, nebst Bemerkungen über Blut und Liquor bei Exazerbationen des paralytischen Prozesses.)** M. PAPPENHEIM, *Monatsschr. f. Psychiat. u. Neurol.*, June 1907, Bd. xxi., S. 536.

IN this interesting article Pappenheim first refers to the well-known facts that leucocytosis occurs in the seizures of general paralysis, and that periods of pyrexia frequently occur apart from these seizures. The pathology of the latter symptom has been much discussed in this connection, and it does not seem settled yet whether its occurrence in general paralysis is due to the inflammatory nature of the lesion or to the fact that this is situate in the central nervous system.

Pappenheim now points out that these pyrexial attacks are accompanied by a polymorphonuclear leucocytosis in both the blood and cerebro-spinal fluid. The findings in the latter were extraordinary, as the number of cells per cubic millimetre was as high as five and six hundred, a number hitherto recorded only in meningitis; nearly all the cells were polymorphonuclear. Five cases are described in full, and the relation of the observations to the pathological condition discussed. ERNEST JONES.

ON THE CONDITION OF THE BLOOD IN GENERAL PARALYSIS
(330) **OF THE INSANE, WITH SPECIAL REFERENCE TO THE CONDITION OF THE WHITE CELLS.** A. E. BODINGTON, *Arch. of Neurol.*, iii., 1907, p. 143.

IN the second stage of general paralysis the polymorphs are reduced below the percentage normal in health. The mononuclear cells are increased. In the third stage of the disease the polymorphs are increased to a percentage much above that normal in health. The mononuclear cells are much diminished. In both stages the large hyaline forms are relatively more abundant than the lymphocytes. Eosinophils are usually numerous in the second stage, scanty in the third. Mast cells are occasion

ally found, and are most numerous when eosinophils are abundant. Myelocytes are occasionally found. There is an evening rise in the total number of white cells in all cases, amounting to from 2000 to 3000 per cubic mm. In the third stage this is associated with a diminished percentage of polymorphs and an increase of lymphocytes.

In a few cases examined for digestion leucocytosis it did not occur.

In the convulsive seizures of general paralysis there is a very marked and sudden increase in the total number of white cells, so that they may be multiplied four to six fold in a few hours. The increase consists of polymorphs. The lymphocytes are reduced in percentage and in actual numbers.

The actual number of hyaline cells is increased. Eosinophils almost entirely disappear, while myelocytes appear in appreciable numbers. These changes may be maintained for one or two weeks, the return to normal being gradual.

The leucocytosis is proportional to the severity and number of the convulsions.

The blood in the third stage of the disease and during convulsions appears dark in colour, viscid, and flows with difficulty. In status epilepticus, while there is a great and sudden increase of white cells, the polymorphs are diminished, the lymphocytes increased. Polymorphs increase and lymphocytes and eosinophils decrease after the fits. Myelocytes are numerous during the status. These changes are transient.

In general paralysis the number of red cells is high; the percentage of hæmoglobin and the specific gravity are reduced.

The blood pressure is low in conditions of mental excitement and in the third stage of the disease, and high in conditions of mental depression. ALEXANDER GOODALL.

ON CLINICAL PICTURES SIMILAR TO GENERAL PARALYSIS.

(331) (**Ueber paralyseähnliche Krankheitsbilder**). J. FINCKH (of Tübingen), *Centralbl. f. Nervenh. u. Psych.*, i., April 15, 1907.

Two cases are reported in considerable detail. The first patient, born in 1848, syphilitic since 1878, came under observation in 1892, complaining of attacks of dizziness and of twitching, to which he had been subject for years. The pupils were unequal, reaction was defective, speech was stumbling, gait ataxic, knee-jerks much exaggerated; he was talkative, euphoric, uttered grandiose ideas, had no insight into his general impairment. The condition of the patient showed no tendency to progression,

and in 1907 he presented essentially the same features, without any additional mental defects, and was more alert and active than on his admission; the memory and retention, which, on admission, were very defective, were excellent in 1907. The stationary condition of the dementia, which consisted in narrowing of the patient's horizon, in the acceptance of hospital life, in exaggerated boastfulness and the absence of the usual defects in memory and retention, led the author to reject the diagnosis of dementia paralytica, which was so strongly suggested by the other clinical features mentioned.

He discusses the possibility of dementia præcox, post-traumatic dementia, arterio-sclerotic dementia, senile dementia, alcoholic pseudo-paralysis, and concludes that the case is one of diffuse brain-syphilis, and suggests that the anatomical basis is syphilitic endarteritis of the smaller vessels (Nissl's form). In laying so much weight upon the nature of the dementia as a diagnostic point, he perhaps fails to do justice to the wide possibilities of dementia paralytica.

The second patient, born in 1852, syphilitic since 1876, showed in 1882 dilatation of the left pupil, and in 1885 mental symptoms; he became irritable, forgetful, inefficient, dull and sleepy, indecent, hypochondriacal. The dilated left pupil reacted sluggishly, the left facial was weak, speech slow, writing clumsy and tremulous, knee-jerks exaggerated, gait spastic. During the seven years of observation he showed considerable variation between periods of dulness and irritable depression, and others of euphoria, with the formation of grandiose plans; auditory hallucinations were a frequent symptom in the course of the disease. In 1887 he had a severe febrile disease which lasted for months. In 1893 he still showed marked mental symptoms, periods of hypochondriacal complaints alternating with absurd boastfulness; hallucinations still persisted. He then left the hospital, and later took an official position which, for the last ten years, he has filled adequately.

After a discussion of the various diagnostic possibilities, the author concludes that the case is one of cerebral syphilis. He admits that as a rule the nervous symptoms of brain syphilis manifest themselves at an early date after the infection, while in the two cases reported a period of eight and ten years respectively had elapsed.

In the absence of anatomical confirmation of the diagnoses the two interesting cases can unfortunately not be used for the purpose of more accurate clinical differentiation of the various forms of dementia. C. MACFIE CAMPBELL.

CATATONIC SYMPTOMS IN THE COURSE OF GENERAL
(332) **PARALYSIS. (Des symptomes catatoniques au cours de la paralysie générale.)** J. SÉGLAS (of Bicêtre), *Nouv. Icon. de la Salp.*, Jan.-Feb. 1907.

THE symptomatology of catatonic states is complex, and embraces, in addition to the general disorder, the more special symptoms of negativism, catalepsy, echolalia, echopraxia, stereotyped gestures, attitudes and utterances.

Catatonic symptoms may occur after the general paralysis is well established, or they may occur at an early stage and make the diagnosis difficult. Cataleptiform traits are much rarer than the phenomena of negativism.

Séglas gives briefly eleven observations to illustrate the presence of these various symptoms in the course of general paralysis.

C. MACFIE CAMPBELL.

ALCOHOLIC PSEUDO-PARALYSIS AND GENERAL PARALYSIS
(333) **DUE TO ALCOHOL. (Pseudoparalisi alcoolica e paralisi generale da alcool.)** G. SAIZ (of Brescia), *Riv. di Pat. nerv. e ment.*, Feb. 1907.

ALCOHOLIC pseudo-paralysis is a non-progressive form of dementia, accompanied by physical signs which sometimes make the diagnosis from general paralysis difficult; abstinence leads to improvement or at least to the cessation of the morbid process. In addition to this form of alcoholic dementia the author considers that a typical progressive general paralysis may be the result of alcoholic abuse in non-syphilitic persons. As the parasyphilitic general paralysis is progressive, so too is the paratoxic general paralysis of alcoholic origin.

He reports briefly eight cases of general paralysis of supposedly alcoholic origin; no mention is made as to how syphilitic infection was excluded, and in none is there any note of the condition of the cerebro-spinal fluid. Such observations obviously prove nothing.

C. MACFIE CAMPBELL.

A CASE CONTRIBUTION TO THE QUESTION OF THE CHRONIC
(334) **ALCOHOLIC "HALLUCINOSIS." (Casuistischer Beitrag zur Frage der chronischen Alkoholhalluzinosis.)** F. CHOTZEN (of Breslau), *Centralbl. f. Nervenh. u. Psych.*, Jan. 1, 1907.

THE question of the chronic alcoholic psychoses is in a very confused state, and the views of the authors as to the chronic hallucinatory conditions in alcoholics are divergent. Several

authors do not admit the existence of a chronic hallucinatory condition as a termination of the typical acute hallucinatory psychosis of the alcoholic. Kraepelin has described a group of cases under the heading of hallucinatory dementia of the alcoholic, but the group appears somewhat heterogeneous.

The author gives the record of a case which demonstrates that the acute hallucinatory psychosis may pass into a chronic hallucinatory condition without any marked mental enfeeblement.

The patient was a man of thirty-six, who had previously had delirium tremens, peripheral neuritis, optic neuritis. Some time after his recovery from the last affection he suddenly developed a typical acute alcoholic hallucinatory condition. Instead of clearing up, the psychosis became chronic, and after two years the hallucinations still persisted; there was a tendency to systematise the ideas of persecution, and some ideas of greatness. The patient was able to work and earn his living, had a fair grasp of general conditions and of the bearing of his illness on his work, talked freely on indifferent topics, but in an involved and evasive manner when questioned as to his hallucinations. During the course of the illness he had a few transitory delirious episodes.

The author justifies in detail his opinion that the whole psychosis was of one piece, and did not owe its special course to any complicating factor either in the way of an original psychopathic disposition or of the development of a dementia præcox process. C. MACFIE CAMPBELL.

PARANOIA: ITS VARIETIES AND MEDICO-LEGAL IMPORT-
(335) **ANCE.** C. BURR (of Philadelphia), *Journ. of Am. Med. Ass.*, June 1, 1907.

THE author refers to psychiatry as the most backward of "sciences." At the present time the classification of insanity is somewhat inaccurate, and the attempts made by the two divergent schools to simplify matters have led to temporary confusion. However, it is generally admitted that all forms of mental disease may be divided into two great classes: (1) accidental insanities; (2) degenerative insanities. Paranoia is a good example of the degenerative type. Ancestral defect is one of the most important factors to be considered in the etiology. Egotism, persecutory and expansive systematised delusions, perversion of moral sense, weak intelligence, falsification of memory, and hallucination are among the most common symptoms. The mode of development and gradual evolution of symptoms is of more importance than the consideration of individual symptoms. Paranoia never shortens life; none recover; dementia is the termination of the disease.

The differential diagnosis must be made from so-called secondary paranoia, which occurs in the course of alcoholism, senility, and in one form of adolescent insanity. Early dementia and hallucinations are important in distinguishing such cases from true or primary paranoia. The disease is of some interest medico-legally, but, compared with the total number of insane, crime is not frequent among paranoiacs.

In court the physician's duty should be to determine the mental condition of the patient; the judge and jury should decide the question of competency or incompetency, responsibility or irresponsibility. C. H. HOLMES.

CONTRIBUTION TO THE SUBJECT OF MELANCHOLIA. (Zur (336) **Kenntnis der Melancholie.**) S. FAUSER (of Stuttgart), *Centralbl. f. Nerv. u. Psych.*, Nov. 15, 1906.

FAUSER holds that certain cases of depression in early life present features practically the same as those of Kraepelin's melancholia of the climacteric period. These cases do not show the retardation and sadness of the manic-depressive group, but present essentially the picture of an anxious and agitated depression. He holds that in these, as in the latter cases, a prodromal period of obsessions is present, and that the disorder may be limited to this stage, and not pass on to the formation of delusions. The suggestion is made to group the early cases of agitation and anxiety which, as a matter of fact, have a favourable prognosis, with those cases of the climacteric melancholia which do not show features of deterioration.

The more severe cases of climacteric melancholia would then be grouped with the other forms of senile dementia, and would simply form a sub-group of the latter, which would really be divided like dementia paralytica into the demented, depressive, expansive, and agitated forms. C. MACFIE CAMPBELL.

ON JUNG'S "PSYCHOLOGY OF DEMENTIA PRÆCOX" AND (337) **THE APPLICATION OF FREUD'S PRINCIPLES OF INVESTIGATION IN PSYCHOPATHOLOGY.** (**Ueber Jung's "Psychologie der Dementia praecox" und die Anwendung Freud'scher Forschungsmaximen in der Psychopathologie.**) M. ISSERLIN (of Munich), *Centralbl. f. Nerv. u. Psych.*, May 1, 1907.

THE views on Dementia Præcox put forth by Jung in his book are so radically at variance with those of Kraepelin's school, that criticism from that side is naturally rather unsympathetic. Isserlin

first gives an abstract of the book itself (for a review, vide *Rev. of Neur. and Psych.*, May 1907), and then enters upon a discussion, first of Jung's method and results, and secondly of the general psychological basis of his presentation of the tissues.

He criticises severely Jung and Freud for their endeavour to give a scientific title to a method which requires so much subjective interpretation as the psycho-analytic method. The method which Freud bases his results upon is obviously an instrument requiring much tact and open to many objections; but Isserlin does not discuss the important question whether the actual cases published by Freud and his followers do not entitle the method to recognition. He does not endeavour to show that the same valuable results, both diagnostic and therapeutic, could have been obtained by some other method. He criticises various points in Jung's studies on association, and holds that the latter has not considered the complexity of the factors which modify reaction-time. As to the general psychological question, Isserlin does not admit that Jung's psychology presents a more living picture of the individual's mental life than the current psychology of the schools; the somewhat artificial language which Jung has adopted occupies his attention, and he sticks at the accidental form rather than discusses the vital truth of Jung's presentation.

C. MACFIE CAMPBELL.

THE IMPORTANCE OF FREUD'S PSYCHO-ANALYTIC METHOD.

(338) **(Die Bedeutung der psychoanalytischen Methode nach Freud.)** SADGER (of Vienna), *Centralbl. f. Nerv. u. Psych.*, Jan. 15, 1907.

THE author, on the basis of his own experience, endorses the views of Freud with regard to hysteria and the neuroses. He asserts that Freud was the first to correctly interpret hysterical symptoms and to indicate the rational method of treatment. The importance of the sexual element in the disorder is given the greatest emphasis: "behind each symptom of hysteria and of the obsession-neurosis lie a number of submerged sexual wishes." Those who criticise such a sweeping statement are met with Freud's charge that they do not have a sufficient grasp of the psycho-analytic technique. Sadger uses as a criterion of one's grasp of the technique the following principle: "Only that man has a mastery of the technique who can trace each individual symptom back to the first four years of life, and not infrequently to the first year." The dilemma is obvious. Sadger communicates no cases, so that the exact value of his support cannot be estimated.

C. MACFIE CAMPBELL.

Reviews

EPILEPSY, A STUDY OF THE IDIOPATHIC DISEASE. By W. A TURNER, M.D., F.R.C.P., London, 1907. (Macmillan & Co.)

IN this work the author has given a careful and thorough account of epilepsy in its various forms. He has reviewed the most important work of other observers, and has added the results of his own valuable observations on the disease and its treatment, an affection which Dr Turner has had excellent opportunities of studying at the National Hospital for the Paralysed and Epileptic, and at the colony for epileptics, Chalfont-St-Peter.

As regards the etiology, the author believes that a "neuropathic predisposition" is the great predisposing cause. Three-quarters of the cases arise under 21 years of age. In those who have inherited the epileptic tendency, a "convulsive habit" may be established, either in the course of natural development, or as a result of certain occasional or accidental causes. The various determining causes of epileptic attacks are considered in detail.

Seventy pages are devoted to a careful clinical study of epilepsy and its complications. Statistics show that the disease terminates in recovery in only 10 per cent. of the cases; in many cases, mental affections, dementia, etc., develop in course of time. Turner's statistics of 150 cases of confirmed epilepsy at the Chalfont Colony show that 2·6 per cent. ended in recovery; 5·3 per cent. retained their normal mental condition; 1·3 per cent. died as a result of the fits; in the other cases mental defects of various forms developed. In 77 per cent. mental impairment of a greater or less degree developed, and 13 per cent. passed into a state of delusional insanity. The statistics as to the cause of death, based on the cases at the Craig Colony, show that 5 per cent. died as the result of a fit, through asphyxia, owing to the patient rolling over on the face, or through food entering the air passages; 12 per cent. died from accident during a fit; 23 per cent. from the status epilepticus; 60 per cent. from intercurrent diseases and other causes.

The pathological changes found in epilepsy are carefully considered. The author regards the following as the more important. The *meninges* generally show slight fibroid thickening. There an increase of the *neuroglia* of the brain, especially in the outer layer of the cerebral cortex. The *nerve cells* are usually diminished in number, probably in direct proportion to the degree of

dementia. In the Betz cells and larger pyramidal cells, changes are found similar to those seen at an early stage of the *reaction à distance*. The cerebellar membranes are often more affected than the cerebral. The glial changes are distributed unevenly over the whole surface of the cerebrum, from the frontal to the occipital lobes. Atrophied and sclerosed convolutions are met with in various regions. The most frequent site is at the anterior end of the cornu Ammonis. The next most usual site is probably in the occipital lobe. Hyaline spheres or lobulated bodies, lying free in the lumen of vessels, and hyaline casts within the vessels are numerous.

The changes in the blood, urine, and cerebro-spinal fluid are considered, and the author thinks that there appears to be proof that some types of epileptic paroxysm may be due to auto-intoxication in persons hereditarily and structurally predisposed to convulsions.

A most interesting chapter of the book (from the standpoint of the general physician and medical practitioner) is that devoted to prognosis. The influence of family history, age of onset, duration of malady, character of seizures, and frequency of attacks are considered carefully as regards prognosis. The author thinks a cure may be expected in 10 to 12 per cent. of the cases. (By "cure" is meant an arrest of the fits for 8 or 9 years.)

The chapter on treatment is a valuable one. The results of prolonged bromide treatment, in 366 cases at the Queen Square Hospital (London), are given. In 23 per cent. the fits became arrested, in 28·7 per cent. improvement occurred.

Since 50 per cent. of the cases derive benefit from the bromide treatment, all cases, especially those of recent origin, should be at first treated with the drug. Turner thinks it is the mode of administration, rather than the drug itself, which is the cause of the bad symptoms. Hence he carefully describes the best method of prescribing the drug. He thinks that the daily dose of 30 to 60 grains is sufficient, and if this dose is not of service, then other treatment should be employed.

If the attacks occur at night only, 30 grains of potassium or sodium bromide should be taken in a small tumbler of water at bed-time. If the attacks occur in the morning, the full dose of bromide should be taken at night, and 15 grains when the patient awakes. If the attacks occur during the day, 30 grains should be taken both at bed-time and after breakfast; or 15 grains at the latter time. In this way 45 or 60 grains of bromide may be continued for a prolonged period. The author states—"Long experience in the use of bromides has taught me that the best and most satisfactory time for their administrations is at bed-time." The omission of the bromides for one day per week is often accompanied by satis-

factory results. Of the bromide salts, those of potassium and sodium are the most efficacious.

The bromide may be given alone, in water, or with arsenic, nux vomica, or gentian. The taste may be partially obscured by camphor, chloroform, or peppermint-water. The syrup of Virginian prune may be added as a pleasant medium for their administration.

The addition of a little digitalis is of service when the arterial tension is low and the heart's action irregular. Chloral hydrate may be added with advantage in cases of prolonged serial epilepsy or of the status epilepticus. The author recommends the continuation of bromide for a long period (5 years) after the arrest of the fits.

In order to test the value of a 30 grain dose of bromide administered at night to a number of confirmed epileptics in an institution, the total number of fits was recorded for one month whilst the patients were taking the bromide, and then for one month when no drug was given: in the first period the total number of fits was 278, in the second period 402. A combination of bromide and belladonna should be tried if bromide alone fails. The author states that his own experience of borax alone and of Flechsig's treatment has been unsatisfactory.

In the status epilepticus, inhalation of chloroform is recommended in severe cases; in less severe forms, 20 grains of bromide and 10 grains of chloral hydrate every two or three hours may be of service. Hydrobromate of hyoscine has occasionally been useful.

We have quoted these details as to the treatment of epilepsy, because they appear of much practical importance.

The author adds his experience on the withdrawal of salt (sodium chloride) from the diet, and on the action of a purin-free diet. Both methods of treatment were of service, a smaller dose of bromide being then efficacious. The major forms of convulsions were most favourably influenced.

The method of education suitable for epileptic children, the treatment of patients in epileptic colonies, and the surgical treatment of the disease are carefully considered; and a useful section is added on epilepsy and life assurance.

Dr Turner's book gives evidence of a most careful and thorough study of epilepsy and its treatment. There is little of importance to criticise. In one respect, however, the work appears to be a little indefinite, that is with reference to the treatment of minor epilepsy. It is well known that this is usually very unsatisfactory, and most readers would have been glad to have a more concise account of the author's experience and opinions on this subject.

Dr Turner's book is a thorough and most valuable work.

Physicians and general practitioners are frequently called upon to treat cases in which cure or great improvement is of the greatest importance, owing to the social position of the patient. Also advice as to the mode of life, diet, education, and prognosis are often required; but only too frequently the medical man's own experience is not sufficiently definite for the expression of an opinion on these points. On nearly every question concerning the clinical aspects of the disease a concise and carefully considered opinion, based on the author's experience and on the published experience of others, will be found in Dr Turner's book. We can recommend the book in the strongest terms. It is the work of a physician who has had exceptionally good opportunities for studying the disease, and the book is a most valuable and important contribution to the literature of the subject.

R. T. WILLIAMSON.

TICS AND THEIR TREATMENT. By HENRY MEIGE and E. FEINDEL, with a Preface by Professor BRISSAUD. Translated and Edited, with a Critical Appendix, by S. A. K. WILSON, M.A., M.B., B.Sc. Pp. 386. London: Sydney Appleton. 1907. Price 9s.

A TRANSLATION of the "Tics and their Treatment" is particularly welcome, since these maladies have not received the attention they deserve at the hands of English writers. The English edition is, however, more than a translation, for Dr Wilson has virtually re-edited the book by introducing various paragraphs from Meige's more recent monograph.

The term "tic" is still so loosely used that Meige and Feindel's insistence upon the necessity for a uniform terminology will meet with universal approval. The authors state that they will be the first to abandon any of the verbal conventions used in the present volume in favour of expressions which, by common consent, may be ultimately regarded as preferable.

A tic is a psychomotor affection, "it is in essence always a perturbation of motility corresponding to a psychical defect." A movement which is provoked by an external cause or an idea becomes a tic when it is carried out without motive. "It is not sufficient that the act be untimely at the moment of execution; we must be persuaded that it no longer stands in any relation to the idea which called it forth at the first, and that its repetition is excessive, its inappositiveness constant, its performance urgent, and its inhibition transient before we can say it is a tic." Brissaud has described a tic as "a pathological habit." Meige and Feindel insist that a particular mental state characterised by defective will power is the essential predisposing factor.

The book opens with an engrossing chapter entitled "The Confessions of a Victim of Tic," which includes many verbatim descriptions from the pen of a *tiqueur*, a man obviously possessed of unusual gifts of self-analysis and descriptive ability. An historical account of the subject precedes chapters which deal with the pathogeny, etiology, and pathological anatomy of tic, the mental condition of tic subjects, a study of the motor reaction, accessory symptoms, the different tics, tics of speech, the evolution of tics, antagonistic gestures, the complications of tics, the relation of tics to other pathological conditions, the distinctive features of tics, their diagnosis, prognosis, and treatment. Re-education is the method *par excellence* from which the most favourable results are to be expected. By the intelligent application of suitable measures "some degree of amelioration is practically always obtainable, and even complete cures may be effected."

Dr Wilson has had the advantage of looking over the proofs of Cruchet's monograph on "Les Torticolis Spasmodiques," a volume of 800 pages which is still in the press, and in a short appendix he indicates certain points upon which the views of Cruchet and some other modern writers differ from those expressed in the book under review.

A very complete bibliography is appended, which cannot fail to be of great value to those who are interested in the subject.

The translation has been admirably carried out, indeed we never remember reading a translation from the French language in which the niceties of expression were more happily preserved.

EDWIN BRAMWELL.

LA RESPONSABILITÉ (CRIMINAL RESPONSIBILITY). (Étude psycho-physiologique. Travaux et Mémoires de Montpellier.) Série scientifique, II. A. MAIRET, Professor of Mental and Nervous Diseases, Univ. Montpellier. Pp. 136. Montpellier: Coulet et Fils; Paris: Masson et Cie, 1907. Price 3 fr.

THIS small book is, its author states, merely an *aperçu* of the subject of criminal responsibility, with which he intends to deal exhaustively in a larger treatise at present in course of preparation. It is ostensibly a study from the physiological and psychological points of view of the intellectual and affective factors of responsibility, and the bearing of the results of their analysis upon questions such as free-will, the innateness of our conceptions of good and evil, and, in particular, the springs of criminal conduct; but it is much more a defence of the author's position—which will doubtless be elaborated in the forthcoming volume—viz., that the determination of the responsibility and the degree of responsibility of an

accused person is a matter for medical experts. This contention is, of course, at variance with the views of jurists and many, probably most, writers on this branch of forensic medicine, who maintain that the sole task of the medical man is to furnish evidence bearing upon the presence or absence of mental disease or disorder in an accused person, and that responsibility is strictly a legal question to be settled by the judge and jury. This latter opinion has for long prevailed in every country, and, with certain differences in the different countries, proof of insanity is required to exonerate an accused person from blame. In every country, however, step by step with the advances made in psychiatry there has been a tendency to extend the application of exonerative clauses. This is, however, not the radical alteration in principle and procedure for which Professor Mairet pleads. Responsibility, he says, is regarded as a function of free-will, of moral liberty, that is to say, of a faculty which does not pertain to science. This, he says, is just; that is, if free-will be regarded in its essence, but not so if it be considered in its manner of expression in action. Outside material things there exist forces—mind, soul, vital principle, free-will, etc.—which science can neither demonstrate nor deny, and which belong to the speculative domain of metaphysics; but what science does prove is that, if these forces exist, they manifest themselves only when their actions are combined with cerebral psycho-chemical actions. "If, then, responsibility exist, it is intimately tied to cerebral function, its study is of a biological order, and in consequence appertains to medicine."

It is evident from the above that the meaning attached to "responsibility" by Professor Mairet is entirely different from that given to it by most jurists and alienists, *e.g.*, Dr Mercier, who says, "responsibility is, therefore, not a quality of the person who has inflicted the pain, but a demand on the part of others that he shall suffer." If, as Professor Mairet maintains, responsibility be inherent in the individual—made up of a knowledge of the moral value of the act, intelligence, fear of punishment, moral sensibility, will, etc.—the determination of whether and how far any individual is responsible must be a matter for the alienist, but if "responsibility" should rightly be interpreted as "imputability," as Dr Mercier affirms, its settlement lies outside medical decision.

R. CUNYNGHAM BROWN.

ARTHUR SCHOPENHAUER. Professor W. EBSTEIN. Pp. 39. Stuttgart: Ferdinand Enke, 1907. Price M. 1.20.

IN this interesting and sympathetic pamphlet Ebstein discusses the medical history of the great pessimist philosopher. His father, who was of an eccentric disposition, probably committed suicide.

His mother, from whom he inherited his literary gifts, belonged to a psychopathic family. Ebstein, however, dismisses the charge of insanity brought against him by Von Sedlitz by showing that his mental life, in spite of numerous eccentricities, had nothing to do with a morbid condition of the brain. Though not quoted by Ebstein, it may be mentioned that Lombroso, in his work "The Man of Genius," instances Schopenhauer as the most complete type of insanity in genius. The longest and most interesting chapter is that devoted to the consideration of the question whether Schopenhauer had syphilis. This charge, which has been urged with great ability by Iwan Bloch, rests mainly on the fact that some posthumous papers show that in 1823 he was undergoing mercurial treatment for some months, both internally and by inunction, associated with tincture of guiacum, which in those days was in vogue as an antisyphilitic drug. The well-known sensual propensities of the philosopher made it seem highly probable to Bloch that he was often exposed to infection, and that syphilis was the chief cause of his pessimism. Ebstein disposes of this argument by showing that Schopenhauer had been a pessimist from his youth up. In this connection it is well to state another objection to Bloch's theory which is not mentioned by Ebstein, but was recently brought forward by Wolf (*Münch. med. Woch.*, 1906, p. 1818), to the effect that the first volume of Schopenhauer's great work, "Die Welt als Wille und Vorstellung," was published in 1818. This fact alone excludes the possibility of a disease which occurred five years later having had any influence on the development of Schopenhauer's philosophy. Further, Ebstein shows that mercurial treatment, especially in 1823, by no means implied that the disease was necessarily syphilis. Mercury, both internally and by inunction, together with guiacum, was strongly recommended by Rust, a celebrated medical contemporary of Schopenhauer, as a remedy for joint-affections, from which it appears Schopenhauer suffered. The nature of this joint trouble is not known. In one of his letters Schopenhauer says he was suffering from gout, hæmorrhoids, and fistula, but Ebstein does not think that there is sufficient evidence to rank Schopenhauer among other celebrated men who have been the victims of gout. His last illness, which carried him off at 72, was probably sclerosis of the coronary arteries, but before its onset his work had been done. Ebstein concludes that Schopenhauer was endowed with a healthy constitution, and that no disease from which he may have suffered had any influence upon his mental work or accounted for the eccentricities of his character. J. D. ROLLESTON.

Proceedings of Societies

AT a stated meeting of the New York Psychiatrical Society, Dr Pearce Bailey read a paper entitled "Recommendations concerning the Improvement of Medico-Legal Methods." It was not difficult to point out glaring defects in our Code of Criminal Procedure, judged by the standards of modern psychiatry, but legal, judicial, and even medical obstacles stood in the way of remedying these defects. Any change from existing conditions would be possible only after a severe and prolonged contest by concerted forces. The fundamental error in procedure in criminal lunacy cases was the test of responsibility, *i.e.* the knowledge of right and wrong as to the act at the time of its commission. This had been condemned by medical writers as being no test at all. Should the letter of the law be strictly observed, no lunatic could under it escape the full punishment for his offence, unless it could be shown that, at the time of the commission of the act, he suffered from distinct clouding of consciousness. Under this law, nevertheless, many murderers had been acquitted by reason of insanity. A loop-hole had been left in certain States—not New York—by adding the "uncontrollable impulse" clause. When this clause does not exist, the fate of the prisoner depends upon the construction which the jury see fit to place on the knowledge of right and wrong as mentioned in the law. The question would naturally arise why, if so unsatisfactory, this test had stood so long with so little variation. The reasons were evident. Defective as is the knowledge of right and wrong as a test of responsibility, no better substitute had ever been offered. Another reason why this law had stood might be found in the fact that the matter is always in the hands of laymen to whom the science of psychiatry is practically unknown. This test would have to endure so long as the Code of Criminal Procedure remained what it is to-day, for the reason that this test is as good as any single test of insanity could be. Modern psychiatry demanded that this test be not replaced by another, but be done away with altogether. No criminal procedure could be at harmony with modern views of mental disease as long as there was no middle course between responsibility and irresponsibility, and no means of judging between them except those furnished by the knowledge of right and wrong. Nothing had been more plainly taught by recent advances in psychiatry than that different mental states make different degrees of responsibility. No law which failed to take cognisance of this fact could be satisfactory to

alienists. Before undertaking any legislation in the line of amending the Code of Criminal Procedure, the Society should bend its energies toward the passage of a law which emanated from the Society and which was in the hands of the Committee at Albany. This law, if passed, would do away with many of the present evils of Court procedure. It was suggested that the model plan for procedure in criminal cases would be that the question of lunacy be referred by the Court to a committee of three, two members at least of which should be medical men. These men should have been certified as to their moral character, and should have passed an examination as to their qualifications to pronounce on the mental conditions. There should be several such experts appointed in every judicial district. The Commission thus appointed should examine into the mental state of the prisoner and report to the Court. They should determine the degree of responsibility—that is, they should determine the character of the mental disease, if any, from which the defendant was suffering, and report in accordance therewith that he was fully responsible, partially responsible, or irresponsible. Partial responsibility in capital cases should be sufficient to remit the death sentence, but no one who had committed murder and who was acquitted on the ground of partial responsibility should be restored to liberty under ten years. In other felonies the cases of partial responsibility should have the shorter sentence optional with the judge. The report of the committee should be handed to the Court and become part of the evidence, and should be the only medical expert evidence.

Dr William Hirsch, of New York City, was of the opinion that no law, however good, would remedy the present condition of affairs so long as we have not to deal with honest men. If it be possible for corruption or any kind of dishonesty to enter legal procedure, then the best law could not be carried out in an ideal way. On the other hand, laws, no matter how defective, could be so handled that they would answer the purposes of humanity and truth if we have to deal with honest men. This Society should direct its attention to its own profession, and endeavour to raise the standard of expert testimony to such a point that dishonesty and incapacity would not be brought to bear in criminal procedures.

Dr William Maben, of New York City, thought the present law unsatisfactory, but so many questions were involved that it would be difficult to formulate anything that would improve conditions as they exist. He advocated referring the matter to a committee, who should investigate conditions not only in this country but abroad. Nothing could be accomplished until the legal profession could be brought into closer touch with the medical profession.

Dr A. R. Dieffendorf, of Middletown, Conn., asked Dr Bailey to state, in closing the discussion, just what standards physicians would have for determining the degree of responsibility. He believed that neither the legal profession nor the community would permit a commission of alienists to pass upon that matter without knowing the rules which the physicians applied. He asked further if Dr Bailey, in looking up the matter, had been able to determine how the criminal procedure in Great Britain had brought about the remarkable condition to which he referred, viz. that there were just as many men found not guilty of first degree murder on account of insanity as were found guilty and hung.

Dr L. Pierce Clark, of New York City, was heartily in accord with the suggestions made by Dr Bailey, and favoured the appointment of a committee to study the matter very thoroughly. The law in the main was far better than what is in force at the present time, but a proper statute could be worked out only after careful study from a legal as well as from a medical viewpoint.

Dr William L. Russell, of Poughkeepsie, N.Y., was, on the whole, in accord with the suggestions presented in the paper, and believed that a committee might, by prolonged study and the presentation of preliminary reports, bring out a discussion of the whole matter that would be very helpful. He referred to the committee appointed two or three years ago by the American Medico-Psychological Association to confer with the American Bar Association, and thought it would be well for the committee under consideration to confer with this and other similar committees. A special procedure might be instituted to determine the advisability of detaining those who have committed murder or homicide, and who have been acquitted on the ground of partial responsibility.

Dr August Hoch, of White Plains, N.Y., said it was not so much a question of making, or urging the making of, a different law in regard to the matter of responsibility, as it was of urging that a better basis be furnished for forming a medical opinion. In most cases experts would not differ so much about a given case if all had the same facts, and enough facts. The divergence of opinion arose because, in many instances, the experts were working with an utterly insufficient knowledge of the case, so insufficient, very often, that in everyday practice no conscientious psychiatrist would think of forming an opinion upon it.

Dr Allan M'Lane Hamilton, of New York City, thought there should be some *rapprochement* between the medical and legal professions, and to this end suggested that a committee be appointed to confer with the Appellate Division of the Supreme Court with reference to the questions in hand. Dr Bailey had been misinformed concerning English laws. In the question of

murder the facts were left to the jury, which inquired into the matter, the question of insanity coming up later. Expert witnesses were not objected to, but the final decision was left to some representative of the Crown. He agreed with Dr Bailey concerning the ten years' confinement of individuals acquitted of murder on the ground of partial responsibility. In disputed cases, however, the time of confinement need not necessarily be ten years.

Dr Charles L. Dana, of New York City, did not take quite so pessimistic a view of the matter as did Dr Macdonald. The committee, if appointed, should confer with prominent judges and lawyers, particularly criminal lawyers. The question of partial responsibility was an important one, and if all alienists would accept the idea that there is such a thing as partial responsibility, it would have its effect upon the method of procedure. It would be useful for the committee to undertake work along the line of devising a test for insanity which might be accepted by the legal profession.

Dr Adolf Meyer, of New York City, said that in practically all European countries partial responsibility has been recognised in some form. The question was whether this could be so formulated as to be satisfactory to the community, for, after all, our laws were not absolutely bad. There had been heretofore too much expression of mere opinion on the part of alienists without enough attention paid to the determination of the actual facts. The relative looseness in the determination of facts and the too liberal acceptation of hypothetical questions on the part of physicians had led to the unfavourable impressions existing to-day. It was extremely difficult to form an idea as to what should be considered sufficient facts to justify an expert in giving an opinion. Adequate observation of the persons should be in some manner invited, if not enforced, and alienists should raise among themselves the standard as to what facts are necessary to justify an opinion. It was the work of the physician to determine whether the individual had any mental disorder, and whether this disorder had anything to do with the commission of the act. The question of responsibility should be decided by the physician.

Dr Pearce Bailey, of New York City, in closing the discussion, said the question of responsibility should be referred to medical men, but they would have no authority more than communicating their findings to the jury. While it might be impossible to change the present procedure, yet it was well enough to look into the matter to see what could be done.

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Review
of
Neurology and Psychiatry

Original Articles

OBSERVATIONS ON THE TREATMENT OF GENERAL PARALYSIS AND TABES DORSALIS BY VACCINES AND ANTI-SERA.

By W. FORD ROBERTSON, M.D., and DOUGLAS M'RAE, M.B., C.M., M.R.C.P.Ed.

THE investigations that have led us to employ specific vaccines and anti-sera in the treatment of general paralysis and tabes dorsalis have been the subject of several previous papers, the last of which formed the basis of a discussion at a meeting of the Medico-Psychological Association in London on the 16th of May. It is necessary, however, to state here in a few words the more immediate reasons, which seemed to us some eighteen months ago to justify the undertaking of these experimental observations of therapeutic aim. Prior to their initiation we had definitely ascertained that a threading diphtheroid bacillus, isolated from the brain and the bronchus of a rapidly progressing case of general paralysis, was capable of producing a subacute disease in rats in which the symptoms were distinctly comparable to those of general paralysis, that on post-mortem examination of the animals, the brain showed the characteristic changes of general paralysis, and that there was also the characteristic invasion by the thread form of the bacillus. It was thus evident that among the various species of diphtheroid organisms that can be isolated from a general paralytic, there is at least one capable of producing the disease general paralysis. We had

also ascertained numerous facts which harmonised with this view. We had obtained cultures of a diphtheroid bacillus from the brain of a general paralytic in ten out of twenty-four cases; and whilst attempts to cultivate the organism from the blood and cerebro-spinal fluid of the living patient had, as in the experience of most other observers, been in most instances entirely negative, we had succeeded in obtaining pure growths from the blood in four cases, and from the cerebro-spinal fluid in two. We had also ascertained that diphtheroid bacilli, in a more or less advanced state of disintegration, are frequently recognisable in these fluids, even when a culture cannot be obtained, and that such altered bacilli are almost constantly present in the walls of the inflamed vessels of the brain in cases dying in a congestive attack. We had also evidence of the occurrence of a diphtheroid cystitis in all of several cases of tabes dorsalis examined, and of the existence of a high intra-corpuscular bacteriolytic index to certain diphtheroid bacilli on the part of such patients and of general paralytics, which we interpret as denoting that they were defending themselves against the attack of an organism of this kind.

With such and other evidence before us, and as our aim in these researches was entirely a therapeutic one, we felt justified in having recourse to specific vaccine and serum-therapeutic measures. As there was reason to believe that the bacilli with which we were concerned produced their toxic effects chiefly by means of their endo-toxines, we decided in the first instance to prepare an anti-bacterial serum, similar to that which Wasserman prepared with the Klebs-Löffler bacillus in 1902. After we had begun to immunise sheep, we ascertained, as the result of the application of Gordon's biochemical tests to various strains of diphtheroids isolated from cases of general paralysis, and through further experimental observations, that at least one other species of diphtheroid bacillus is often virulent to mice and rats, and is capable of causing the symptoms and lesions of general paralysis. We therefore employed both of these two types of bacilli in our therapeutic experiments. Both have a close morphological resemblance to the Klebs-Löffler bacillus. The first, which we have designated the *bacillus paralyticans longus*, differs from the bacillus of acute diphtheria in producing no acid in the control broth in Gordon's bio-chemical tests, in being non-virulent

to guinea-pigs, but occasionally virulent to mice and rats, and in forming threads under certain conditions of growth. The second, which we have termed the *bacillus paralyticans brevis*, is distinguished especially by its bio-chemical reactions; it produces acid abundantly in saccharose and glucose broths, but forms no acid in lactose, salicin, starch, dextrin, glycerine, and control broths; it is not virulent to guinea-pigs, but is often very virulent to mice and rats. We believe that these are not the only species of diphtheroid bacilli that are commonly concerned in the production of the toxæmia of general paralysis and tabes dorsalis, but that, just as in the case of the pathogenic streptococci, numerous different but closely allied species are capable of exercising a similar pathogenic action.

Method of Vaccination.

The material employed was a suspension of a weighed quantity (10 to 30 mgr.) of the bacilli in 2.5 c.c. of sterile saline solution heated to 60° C. for 15 minutes.

The skin of the limb was cleansed by rubbing with a 1 per cent. lysol solution, and injections of the emulsion were given hypodermically. Local effects varied from the merest induration at the site of puncture to erythema and œdema of the subcutaneous tissues.

The immediate general effects were the production of brief pyrexia, flushing of face, headache and drowsiness, while increase of tremors, ataxia, and a return of lightning pains occurred in some cases. Mentally, there were remarkable changes in not a few of the cases.

Case 1. Female, a third-stage, bed-ridden general paralytic, with contractures and lightning pains of an intensely distressing character. Three injections were given and the pains were increased for a time after each. Three weeks after this she began to make active efforts at dressing herself and walking about with the aid of a chair, and was able to express herself coherently, while the pains seldom recurred. Two months later she was able to scrub floors and go about the wards without support. She was never able to stand erect, however, on account of permanent flexion at the knees. This improvement was

maintained for a year, when the patient was removed from the asylum.

CASE 2. Female, a third-stage general paralytic, very helpless and intensely confused in mind. Three injections gave temperature reactions of over 100° F. each time. Increase of tremors and grosser impairment of speech followed the first inoculation. Shortly after the second the patient talked sensibly and with clear articulation, and she was able to go about. Two months afterwards she relapsed into her former condition.

CASE 3. Female, a stuporose general paralytic in second stage. Three inoculations were followed by rises of temperature to over 100° F., and a temporary lucidity in the patient's mental state was noticed.

CASE 4. Female, a non-progressive general paralytic, the subject of "pains" and "neuralgias," had nausea and vomiting after the first injection and became depressed in mind with exacerbations of pains after the second and third inoculations. Three weeks later she became cheerful and employed herself in house-work for the first time since admission, twelve months previously. She relapsed, however, several weeks later.

CASE 5. Female, a depressed and confused tabo-paralytic, became lucid and gave a correct account of her family history after the first inoculation. After two more inoculations she employed herself in the ward, but relapsed after three weeks to her former state.

CASE 6. Female, was in third stage and bedridden for three years, became excited and impulsive a few hours after injections. She developed diarrhœa and had a marked exacerbation of leucorrhœa. No improvement was noticed.

CASE 7. Female, a confused general paralytic in the second stage, had a return of pains, and became flushed after inoculation on three occasions. Her speech, at first more defective, became for a time clearer than formerly.

CASE 8. Female, an early third-stage general paralytic, became excited and violent after each injection and improved considerably in bodily condition later.

CASE 9 was that of a medical man who had been developing symptoms of tabes for over two years. His reactions to inoculations of the toxins of a diphtheroid bacillus of the "brevis" type, isolated from his bladder, are interesting and

significant. The left arm was injected with 10 mgm. of dead bacilli, and the patient in a few hours suffered from distinct malaise, and had a definite attack of lightning pains for twenty minutes. These had not troubled him for a fortnight previously. There was no rise in leucocytes. Pains recurred on the second and third day. Leucocytes remained about 9000. The second injection of 20 mgm. of bacilli a week later resulted in a rise of temperature to 99° F. at 3 A.M. Next day there was marked local reaction, and he had considerable malaise. There was slight recurrence of "pains" and the leucocytes were 16,000. After the third injection he suffered from severe headache and had return of lightning pains for half an hour, about twelve hours from the time of inoculation. Next day there was a leucocyte count of 17,000.

On the whole the patient was better and had not the same mental depression that accompanied his ailment prior to inoculations. A fourth injection caused very marked malaise, a temperature of 100·2° F., and once more a return of the pains for a short time. Subsequently he expressed himself as being better in health, much longer free from pains, which moreover were considerably diminished in severity.

The results of these observations would seem to show that repeated vaccinations might prove a useful mode of treatment in general paralysis and tabes. This belief is supported by the results of the independent observations of O'Brien[1] in America, who has recently reported that he has obtained considerable success by such methods in cases of general paralysis.

That we have not pursued the method further is entirely on account of the fact that our original object was to prepare a specific anti-serum, and we merely temporised with vaccines until the serum was ready for use.

In our opinion such vaccine treatment would have to be carried out under the following three conditions:—(1) The bacillus ought to be one isolated from the patient; (2) There should be evidence that it is exercising a pathogenic action upon the patient; and (3) The injections should be carried out under the guidance of the leucocyte count or the opsonic index.

[1] *Journ. Am. Med. Assoc.*, June 29, 1907.

Preparation of the Anti-Sera.

Sheep were selected as most convenient for our purpose, and in March 1906, two healthy animals were first of all inoculated with dead cultures of bacilli of the *bacillus paralyticans longus* type, isolated from the brain of a case of general paralysis.

Weighed quantities of the living bacilli were then injected once a week, and the temperature of the sheep was taken twice daily. The inoculations caused a local induration and tenderness at the site of puncture, a rise of temperature of from one to two degrees, and a slightly torpid condition of the animal. After two months, as the injections failed to cause any of these phenomena, and as the intra-corpuscular bacteriolytic index had been raised, it was decided to use the serum. We have now twelve sheep immunised to various strains of virulent diphtheroid organisms, six to the *b. paralyticans longus* type, four to the *brevis* type, and the remaining two animals to both types.

Obtaining the Serum.

The animal is lifted on to a specially contrived stool, the front of the neck shaved and sterilised with 5 per cent. lysol and carbolic lotions. A large syringe-needle with a bore of 2·2 m.m., which has been sterilised in hot oil, is grasped by means of special forceps; the thumb of the left hand, enveloped in a carbolic swab, is pressed against the external jugular vein low down in the neck; then the needle is plunged into the distended vessel. The blood is collected in quantities of 40 c.c. in 60-c.c. glass tubes, previously plugged and sterilised in a hot-air chamber. The serum separates out in twenty-four hours to the amount of 20 c.c. in each tube, though it often takes forty-eight hours to reach this quantity. It is now ready for injection, though it may remain in contact with the clot for a month or so, till required.

Mode of Administration.

The sera have been given by hypodermic injection, by the mouth, through the nose, and, in one case, per rectum.

For hypodermic injection we use an all-metal serum syringe,

which has been thoroughly cleansed in carbolic lotion, and then repeatedly filled and emptied with oil at a temperature of over 160° C., a method employed by Dr Stenhouse Williams of the Runcorn Serum-Farm, Liverpool.

The skin of the abdomen is carefully cleansed with 1 per cent. lysol, and the injection is given with not too small a needle (1 m.m.). The skin at the site of puncture is pinched before and after removal of the needle and held for half a minute in order to seal the wound and prevent any escape. A piece of fresh wool is placed on the spot and the fluid is left to be absorbed.

Given by the mouth the serum is quite palatable if some peppermint water or cassia is added. We also administer it by means of an ordinary glass syringe through the nose, passing 10 c.c. slowly along each nostril, directing the patient to retain the fluid for a little time before swallowing.

Local Effects.

Given hypodermically, the serum may produce no local effects, or merely a little induration with or without erythema, which in a few cases may spread and give rise to œdema and brawny swelling of a large area of the abdominal subcutaneous tissues. This is readily subdued (in 24 to 48 hours) by painting the skin with a solution of picric acid. Some cases are more prone to this reaction than others, and it suggests the local formation of toxins by destruction of bacilli in the tissues. It has been found also that friction of the clothing during active exercise is liable to cause a degree of inflammation if the patient is not kept in bed for the day after injection. Serum urticaria occurred in nearly every case.

When the serum was given by the mouth, local effects were manifested by the development of nausea, vomiting and diarrhœa in some cases, and the occurrence of a feeling of hunger in nearly all cases.

General Effects.

These were chiefly drowsiness, diaphoresis and malaise, whilst polyuria and exacerbation of chronic leucorrhœa were occasionally observed.

Immediately after injection, or in about half an hour in oral

administration, flushing often occurred; some of the patients complained of being giddy with a "tight feeling" in the head and a "stiffness of the face." Some experienced temporary loss of vision. In one case the patient was blind in one eye for ten minutes nearly twenty-four hours after injection. Others asserted they felt as if they were drunk. Vomiting occurred in some cases after hypodermic injection. Mentally, many of the patients became more confused; most of them experienced an unusual sense of well-being, and some were hilariously excited. The speech, gait and co-ordination were for a time more impaired, whilst the tremors were increased, and many of the patients complained of shooting pains and sensations of numbness and tingling or feelings of "pins and needles."

The temperature reaction is characteristic, and so far as our experience goes it is diagnostic. It has for its features a rise to 100° F., or more, when the serum is given hypodermically, and to 99°, or more, when given by the mouth. This rise occurs within twelve hours and is over in twenty-four, although in the case of mouth administration it may be delayed till the following day.

It has been observed that a temperature reaction may fail to occur if the patient is in a state of remission, when the serum is taken close upon the ingestion of food, or if diarrhœa occurs immediately after its administration by the mouth.

The pulse-rate is usually increased, but on account of its erratic character in cases of general paralysis and other forms of mental disease (well seen in a study of the charts of these cases kept for over a year), no useful data can be obtained.

The serum was tested originally in four cases of general paralysis in the final stage of the disease. In addition to a raising of temperature a few hours after injection, a remarkable lucidity of mind temporarily supervened in three cases. One patient, who was in a congestive seizure and comatose, became quite conscious two hours after the injection of 15 c.c. of serum, and the convulsions ceased and did not recur. She remained conscious and lucid up till the time of her death, fifteen days later. Recently another patient, admitted to the asylum while in a congestive attack, who rapidly developed coma, became quite lucid within an hour of the administration of the serum.

The convulsions persisted, however, and she died in two days, but maintained consciousness to the end.

The therapeutic effects on the mental and motor symptoms of the patients treated can best be illustrated by a short summary of a few cases.

Cases Treated.

Case 5 (C. S.), female, æt. 28, married. Admitted in July 1906, in a confused apathetic mental state, being rather fatuous and facile. There was general muscular enfeeblement with paresis and ataxia of gait, Rombergism, absence of knee-jerks, considerable dysarthria and marked labial and lingual tremors. The pupils were very irregular, unequal and sluggish to light and accommodation.

In August she developed a congestive attack, lasting twelve days, during which she had ten severe epileptiform seizures, which left her with flaccid paralysis of the right arm and leg for a few days. A month later she had a similar attack lasting two days, during which a leucorrhœal condition was aggravated.

Serum treatment was begun on the 24th September, and had the usual effects. In January a sensation of "pins and needles," lasting for twenty minutes, was all that occurred to suggest an abortive congestive attack. In February she had so far improved as to be able to attend the weekly dance. Improvement continued up till the end of April, when it was noticed that six consecutive doses of anti-serum gave no temperature reactions. About this time she became markedly constipated, and developed a congestive seizure on the 2nd May, which caused a slight paresis of left side of face after twitching had occurred over both sides of face and in left arm. There was no loss of consciousness, and, being perfectly lucid, the patient was depressed on account of this return of the seizures. On this occasion the temperature, which stood at 101° F., was raised to 103° F. after 20 c.c. of serum had been given by mouth. Three days later she was out of bed and did a hard day's work at "spring cleaning," with no ill effects.

Her present condition (July, 1907) is one of almost complete lucidity, with normal articulation. The pupils react briskly, though they are still unequal and irregular. The facies is

almost quite free from *amimia* and tremors, though the knee-jerks remain absent and the tongue still shows fibrillar twitchings.

CASE 6 (C. M.), female, æt. 48, married. Admitted in June 1906, with obvious symptoms of general paralysis. She became a confused, amnesic, bed-ridden paralytic, who lay huddled up in bed, unable to feed herself or have control of the sphincters.

Serum treatment was begun in September. The usual temperature and other phenomena were observed. Two months later she was up, able to read a book and to converse a little. In January she could knit and do light house work, recognised her relatives and appreciated their visits. This improvement has been maintained.

CASE 7 (Mrs S.), female, æt. 24, married. Admitted in September 1906. Was a bed-ridden, rapidly-progressive general paralytic with large bed-sores. The knees were acutely flexed, there were marked tremors, the speech was inarticulate and unintelligible, and there was complete absence of intelligent response.

Injections were begun on September 24th and were followed by typical and well marked reactions. Two months later the bed-sores healed and she was able to get up for a little. Improvement has continued and she is now actively and intelligently employed in the wards. Beyond a slight degree of facility she betrays at present no mental symptoms of her disease, and the motor signs are limited to fixed pupils and abolition of knee-jerks.

CASE 10 (J. H.), female, æt. 48. Intemperate and dissolute for seven years after the death of her husband. Admitted in July 1906, in a state of excited melancholia with symptoms suggesting peripheral neuritis. Six months later, while in a state of sub-acute mania, she exhibited marked unsteadiness of gait, and lent and lurched to the right side while walking. The articulation became very defective. The pupils were found to be unequal, very irregular in outline, and sluggish and limited in their movements both to light and accommodation. Early in February she was put under serum treatment and gave typical reactions. The day after the first injection the patient expressed herself in a rational way for the first time since her admission,

eight months previously. She steadily improved and passed into a state of complete remission and has technically recovered and been discharged from the asylum. On the day she left, the following motor symptoms were noted to be present :—The pupils were irregular, the right particularly so; both were limited in their range of movement to accommodation, and the right was distinctly sluggish and limited in its response to light; stumbling and occasional slurring of speech, a slight tendency to lurch to one or other side while walking, and tremors of facial and lingual muscles were also to be observed.

CASE 12 (M. A. H. B.), female, æt. 35, married. Admitted in 1900. A case of dementia with the motor symptoms of general paralysis, which had not cleared up under vigorous anti-syphilitic treatment. She had passed gradually into the third stage, had been bed-ridden, resistive, irritable and absolutely mute for over a year. She was unable to feed herself or attend to the calls of nature, and was extremely emaciated. Serum treatment was begun in February of this year and the usual reactions occurred. Within two months she was able to walk in the garden, could feed herself, and had control of the bladder and bowels. She can now express herself coherently, is delighted with her improvement and proud of her ability to do light house-work. She is up and goes about all day, takes an interest in her surroundings, and is sociable and amiable.

Time does not permit of a further individual survey of the cases, but we may here summarise the results obtained up to the present.

Altogether, thirty-four cases of general paralysis have been subjected to the anti-sera, and all of these have yielded a positive result, as regards temperature and other phenomena. Two cases of tabes have also shown similar reactions. With regard to the therapeutic results, out of twelve cases under our own immediate supervision for over a period of three months, all have shown remarkable degrees of improvement. Of nine cases treated indirectly by us for a sufficiently long period to obtain therapeutic results, four have improved up to the present, and two have become well enough to leave hospital and to enjoy ordinary social life, and are considered capable of taking

care of themselves. The remaining three have become progressively worse in spite of the vigorous application of the sera. One of them was in a state of almost complete remission for a time, but relapsed completely while absent from medical supervision.

In the case of the medical man with tabes, the employment of anti-serum prepared with his own particular "diphtheroid" resulted in typical temperature reactions, considerable malaise and an immediate return of lightning pains at first. He has now been under serum treatment for five months. He is satisfied that the severity of the pains is very greatly diminished, while the duration of the periods of remission is increased and he no longer experiences unpleasant paræsthetic sensations as formerly. Similar improvement has occurred in another case of tabes of over twelve years standing, which has been a shorter time under serum treatment. The ataxia in his gait is much less marked. He has regained his facial expression. The pains, which occasionally return about six hours after taking serum and last twelve hours, are much less severe, and he has longer periods of freedom from them than he ever had prior to serum treatment.

Control Cases.

It may here be convenient to refer briefly to eleven cases which were subjected to the anti-sera in order to control our observations in the cases of general paralysis. Three were cases of dementia præcox, four of excited melancholia, two were suffering from alcoholic dementia, one was epileptic and another laboured under mania. They all had the anti-sera on at least three occasions, two cases having them as often as eleven and ten times respectively. In none of these did any specific reaction occur after mouth administration, and any rise of temperature subsequent to injection could readily be explained by other factors.

Dr Alexander Russell, at present assistant physician at Morningside Asylum, made daily observations over a period of four months on the leucocytes of some of the cases treated by serum. He found that in the cases of general paralysis there was no material change in the numbers of leucocytes, or in the differential

counts after injection of the anti-sera; whereas, in control cases, in most instances there was a decided rise in the number of leucocytes, while the polymorpho-nuclear percentage showed a tendency to rise.

Control Sera.

Cases of general paralysis were also subjected to other sera in order to test the specific character of our anti-sera.

Normal serum was obtained from each of the sheep prior to the inoculations with bacilli, and this was injected in doses of 20 c.c. into ten cases of general paralysis on more than one occasion, without the slightest reaction.

Again, nine of the patients were injected each with 10 c.c. of polyvalent anti-streptococcic serum with no reaction traceable to it.

The reactions resulting from the use of our anti-sera could not be confounded with "serum disease," since they do not correspond to it, and they occurred only in the general paralytics. Urticaria, and occasional accidental local inflammation at the site of injection, were the only reactions common to both the general paralytics and the controls.

In conclusion, we would say:—

1. That the anti-sera with which we have been working produce reactions which are diagnostic of general paralysis or tabes dorsalis; they are probably due to the liberation of endo-toxins.

2. Cases of these diseases treated with the sera in most instances undergo improvement.

3. A polyvalent anti-bacterial serum is likely to be more efficacious than either the mono- or bivalent serum we have hitherto used.

4. One of the chief obstacles in the way of obtaining a very potent serum has been the loss of virulence in the strains of the organisms used.

5. There are grounds for believing that an anti-toxic serum would be of use, especially for the immediate treatment of conjestive seizures.

6. Lastly, in view of the presence of dissolving bacilli in the brain of the general paralytic, where in all probability they produce extremely virulent endo-toxins, another aim should be the production of a serum containing chiefly an anti-endo-toxin.

THE PATHOLOGY OF EPIDEMIC CEREBRO-SPINAL MENINGITIS.

(*Continued.*)

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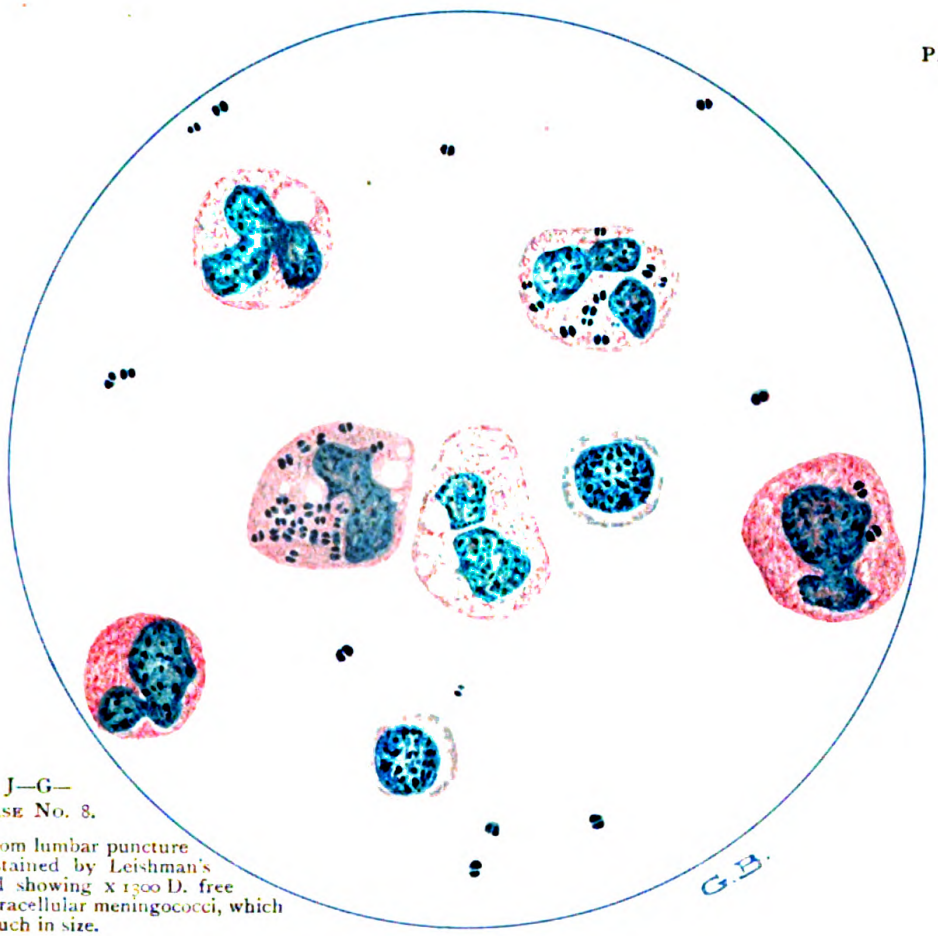
THE MENINGOCOCCUS FROM A MORPHOLOGICAL AND BIOLOGICAL STANDPOINT: BASED ON THE STUDY OF THE ORGANISMS FOUND IN FIFTY CASES.

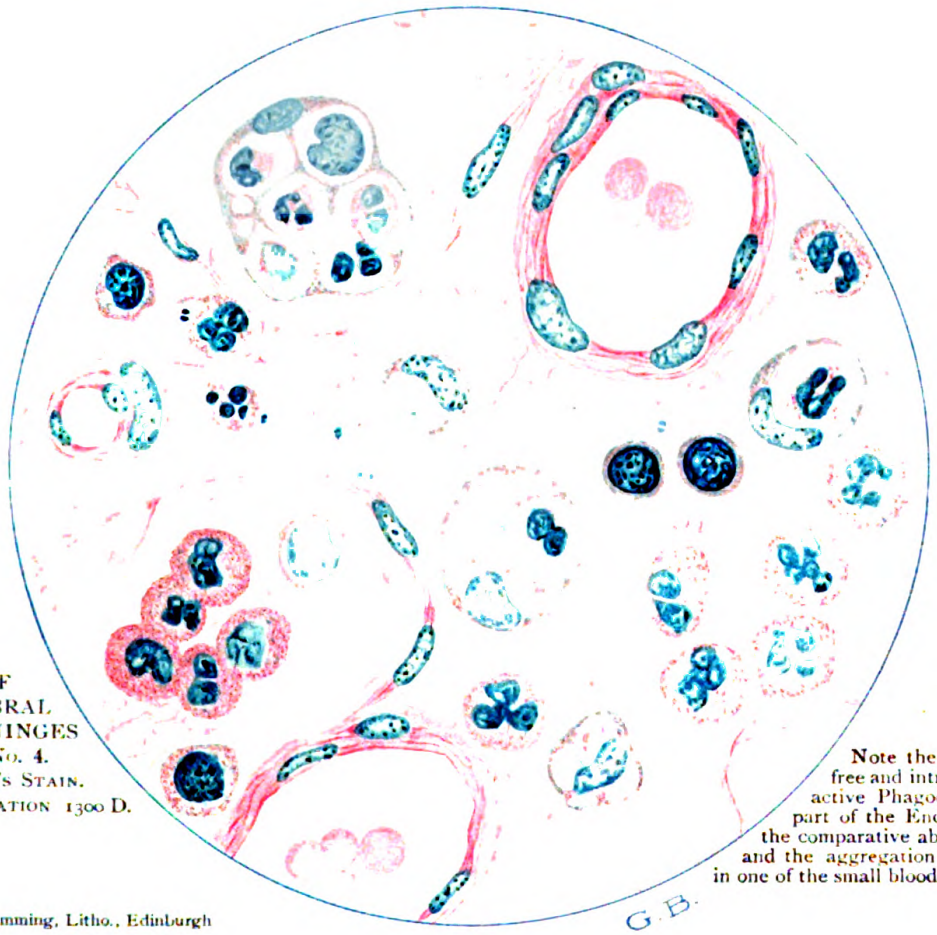
THE meningococcus as seen in the cerebro-spinal fluid is an organism with such definite morphological characters that it may be identified in that situation with a fair degree of certainty by examination of films alone. Absolute identification, especially in situations apart from the central nervous system, requires a much fuller investigation, and is sometimes impossible. As seen in the turbid cerebro-spinal fluid in a case of the disease, the organism has the following characters:—

It occurs as a round or slightly oval coccus, sometimes single, usually seen in pairs as a diplococcus, the opposed surfaces of the cocci being flattened. I have not seen chain forms. The organism is most usually situated within the polymorpho-nuclear leucocytes of the exudate, but free forms are also seen in some cases. These may be due to the disintegration of polymorphs, but I am inclined to the opinion that where the organism is very virulent, the intra-cellular position is not so constant a feature. This certainly seems to be the case where animals are given excessive doses of cultures. The number of the organisms varies much; sometimes even in the most acute cases a prolonged search is necessary to demonstrate even a single pair of organisms, though in the search numerous minute particles are often seen in the cells of the exudate, which one feels morally certain represent degenerated meningococci. At other times the organisms are extremely numerous, and the polymorphs may be crowded with them. The organism shows great variety in size, and in many cases the presence of involu-



J—G—
CASE NO. 8.

Film from lumbar puncture fluid, stained by Leishman's method showing x 1300 D. free and intracellular meningococci, which vary much in size.



ECTION OF
HE CEREBRAL
MENINGES
IN CASE NO. 4.
LEISHMAN'S STAIN.
MAGNIFICATION 1300 D.

Note the meningococci free and intracellular; the active Phagocytosis on the part of the Endothelial cells, the comparative absence of fibrin and the aggregation of leucocytes in one of the small blood vessels.

M'Lagan and Cumming, Litho., Edinburgh

tion forms is a striking feature. These may be pale, oval, or rounded bodies up to and over two micros in diameter. They frequently show irregularity in staining, so that sometimes large oval forms may give the appearance of polar staining in a short oval rod almost like B. pestis; sometimes the staining is concentrated at one side of a round form, giving a signet-ring appearance. Leishman's stain is a satisfactory method for demonstrating the above appearances. A common appearance with this stain also is that of a finely granular detritus among the cells, at first sight suggesting very minute cocci. I am of opinion that this detritus represents the disintegrated remains of meningococci. The diplococci are sometimes seen inside large mononuclear cells, but usually this is only due to the ingestion of an infected polymorph.

The organism stains with any of the ordinary aniline dyes; in the great majority of cases it loses the stain rapidly in Gram's method. I have not yet seen a variety from the tissues which gives a positive reaction to Gram's stain, but I have on several occasions seen organisms in fresh films just prepared from the cerebro-spinal fluid, which after three minutes in the gentian violet solution, and one minute in the iodine solution, resist the decolouring action of absolute alcohol for at least one minute. As Vansteenberghe (27) and other competent observers have described Gram positive forms even from the tissues, the Gram reaction must not be made a conclusive point in the identification of the organism. As regards the question of the organism being capsulated, I am not convinced that a poorly defined capsule does not exist. It is so indistinct, however, that there is no excuse for confusing it with the pneumococcus on this ground. The capsule I have observed is quite different from the appearance due to plasmolysis, which I have also seen. As indicating the supreme value of the direct examination of films from the fluid obtained by lumbar puncture during life, I may mention here that in fifty consecutive cases of acute meningitis I have found the meningococcus present with no other organism forty-seven times. In one case a bacillary form of a leptothrix was found; in another the same leptothrix form, together with a coccus of whose nature I am still in doubt; while in the third the pneumococcus was present by itself.

At the post-mortem examination the organisms have been

demonstrated in several other situations; thus it was seen twice in the knee-joints, twice in the pericardium, and in one case during life it was cultivated from the blood. The blood, however, was only cultivated in three cases. With regard to the presence of the organisms in the lung, I am not able to speak with certainty. It is true that on several occasions Gram negative diplococci having all the morphological appearances of meningococci were seen in this organ; but as the organisms were never present by themselves, but occurred alongside pneumococci, staphylococci, and other organisms, attempts to isolate them invariably failed.

Cultivation of the Organism.

Like other observers, I have found the cultivation of the meningococcus a matter of considerable difficulty. Growth is said to occur at as low a temperature as from 25° to 27°C., but practically speaking there is only growth and multiplication of the organisms at the body temperature. The medium I have found most invariably successful has been ovarian agar, but even on this medium failure has been frequent, though plenty of organisms have been present in the fluid from which the culture was being attempted. This, indeed, may be said to be almost a special feature of the meningococcus, and in the cerebro-spinal fluid, or in exudates artificially produced in the lower animals, apparently only a very minute proportion of the organisms are viable. The meningococcus, however, shows very considerable variations in its capacity for growth on various media; thus, in certain cases I have had no difficulty in obtaining a fairly copious growth on ordinary agar, and the same may be said about Loeffler's blood serum, blood agar, and glycerine agar, The organism is a fairly strict aerobe.

Naked-Eye Appearance of the Colonies.

On ovarian agar, serum agar, etc., colonies may be visible in 14 to 18 hours, but as a rule they are very small, even at the end of 36 hours. I have also seen the appearance of the colonies delayed for 48 hours. I am not of opinion that the appearance of the colonies can be considered of any great importance in the identification of the organism. They may be

readily differentiated of course from the dead white, opaque colonies of staphylococcus albus, which is one of the most common varieties of contamination. As a rule the colonies are rounded with flat, moist, shining surfaces; the margins of the colonies are usually round, but slight indentations may be seen. The periphery of the colonies is generally more clear and transparent than the centres, which are more grey, granular, and slightly opaque. There is often quite a close resemblance to young colonies of B. coli. Occasionally the colonies become confluent; this is, as a rule, more often seen in strains which have been growing for a considerable time apart from the body. Though the above may be considered the typical appearance, one occasionally sees colonies of meningococcus which are much more opaque. On almost any medium the meningococcus degenerates very rapidly, and, at least when recently removed from the body, frequent sub-cultivations have been found necessary in order to keep the culture alive. Involution forms appear with great rapidity, especially if the surface of the medium be dry, and at the end of three days, sometimes even sooner, hardly a healthy organism can be demonstrated in films. Swollen coccal forms staining poorly are the most common in this involution, but oval and slight, elongated forms are also met with. After three or four days little may be seen in film preparations but a granular detritus, though every now and again one sees in this debris an organism staining perfectly well. In a 24- to 36-hour culture on a favourable medium the following is the most common appearance in films.

The organism appears as single cocci; diplococci with the typical flattened opposed surfaces and tetrads are not at all uncommon. Almost from the first very considerable variations may be noted in the size and staining intensity of the cocci.

Staining Re-Actions of the Organism.

On this occasion I propose to refer very briefly to this subject.

Young cultures stain readily with the ordinary aniline dyes, but much difference of opinion exists with regard to the reaction to Gram's strain. In the great majority of the strains I have been dealing with, the organism has been uniformly and

constantly Gram negative; but in the case of three different strains from cases 4, 5, and 8 in my series, I have observed a Gram positive reaction on the part of some elements, at least. A study of the literature since Jäger's original paper convinces one that the meningococcus has been from time to time confused with other organisms which are Gram positive; but admitting this, certain observations remain which convince me that occasionally the meningococcus itself may show a Gram positive reaction. That this is a rare occurrence is evident, for many observers have never seen it, and in my own experience it has been noted only thrice in the examination of over fifty different strains. As may be seen from the résumé in the first part of this paper, several Continental observers have noted this variation on the part of the meningococcus; and Vansteenberghe in particular has described from a fulminant case of cerebro-spinal meningitis an organism which in every way corresponded to the meningococcus, but that it was Gram positive. Reference to one strain which I separated from case 8 may be made here. The organism was Gram negative in the tissues, but in one culture some Gram positive elements appeared. In old cultures (of various organisms) this is not an uncommon appearance; but in my experience at least it is exceptional in a 20-36 hours' culture to see a diplococcus, one coccus retaining the Gram strain, and one losing it completely, as was present in this case. The organism also occurred in the form of short chains of three and four cocci, and in the middle of such a chain one of the elements appeared Gram negative, while the others were Gram positive. These appearances are not what one would expect in the case of a contamination of the original culture by a second organism; but in addition one found that when this culture showing these Gram variable forms was introduced into the peritoneal cavity of a mouse, the animal died just as when true meningococci are inoculated, and in the films from the exudate only typical Gram-negative diplococci were found which culturally and morphologically corresponded to true meningococci. I am therefore convinced that the meningococcus does occasionally show this Gram-positive reaction, and with an organism so variable as we know the meningococcus to be, it is not surprising that this does occur. This strain is also interesting, as it shows that the meningococcus does occur sometimes in the form of short chains, in culture

at least, though Gordon (28) has stated definitely that this never occurs. I have also observed it in other strains where the organisms were constantly Gram negative throughout. I have never seen the long streptococcal forms which Jäger described in his original paper. Though I do not propose to describe the growth of the meningococcus on other media, I may state that on no occasion did I succeed, even in the case of old cultures, in obtaining a growth in Gelatine. In Bouillon, however, growth occurs, but not freely. I have found that growth is more copious if the broth be briskly shaken up at intervals. This acts probably by aerating the medium. After two or three days a slight scum may form on the surface. Growth also occurs in milk, though not freely; faint acidity is produced, but the medium is not coagulated.

During recent years much attention has been directed to the possibility of differentiating micro-organisms, from their varying power of fermenting different sugars with acid production. One is not predisposed in favour of this method, as it is essentially based on very delicate and complex chemical processes, and it is often very difficult to obtain many of these sugars in a chemically pure state. Von Lingelsheim's results (29) in the case of the meningococcus, however, are so uniform that the method is worthy of a full investigation, and many competent observers believe strongly in it.

The subject has also been investigated in this country by Gordon, whose results, however, do not quite correspond with Von Lingelsheim's. The method had grown out of the attempt to distinguish by its means the meningococcus from other diplococci which some observers have found along or together with meningococci in the cerebro-spinal fluid in cases of acute meningitis. To one form has been given the name diplococcus crassus, and it has been supposed that this organism has been confused by Jäger and his followers with the true meningococcus. Von Lingelsheim himself is evidently not quite convinced that the diplococcus crassus has not some connection with meningococcal meningitis, though he describes it as occurring in other conditions as well.

As I have not worked personally at the toxines of the meningococcus, I do not propose to refer to the subject, beyond mentioning that all the observers, *e.g.*, Albrecht and Ghon (20),

who have investigated it, agree that the toxin is of an endocellular nature; in support of this view the same observers were able to produce a similar peritonitis to that seen when the living organisms are used, by intra-peritoneal inoculation in white mice of cultures killed by heat.

Before leaving the subject of the bacteriology of meningococcal meningitis, I should like to refer to the association of the meningococcus with other organisms. There is little doubt that secondary contamination of the cerebro-spinal fluid is frequently present at the post-mortem; and even during life, in the course of a meningococcal meningitis, other organisms may reach the central nervous system. I have seen the pneumococcus together with the meningococcus in one case, and in three cases I have found diphtheroid bacilli; in one of these latter cases I was able to prove by cultures that these were merely bacillary forms of a leptothrix, and though they seemed in two of the cases to have modified the histological changes in the meninges, there was no evidence that they were the essential ætiological factors. In one case, it is true, no other organisms were present, but the meningococci may readily have disappeared. In the other two, diplococci were present; and though I am unable to state definitely that they were meningococci, in one of the cases at least I have little doubt that the case was originally meningococcal. As the pharynx and respiratory tract are the commonest sites of these leptothrix forms, it may be that they have entered by the same channels as the meningococcus, and thus may afford a clue to the real portal of entrance of that organism. In any case the observation is important, as it shows that in a grave, acute disease of the central nervous system, secondary invasion by other organisms may occur; and as the real, exciting agent may disappear, these secondary invading organisms may be mistaken for the real causal organisms.

Experimental Observations on the Meningococcus.

As the lower animals are relatively insusceptible to the meningococcus, considerable difficulty has been met with in undertaking experimental work with this organism; and until Flexner (35) obtained his results in monkeys, no substantial advance has been made in this direction.

Like others, I have found that intra-peritoneal inoculation in mice gives the most satisfactory results, but even here very large doses of cultures must be given, and occasionally the inoculations fail. Generally the animals die of an acute toxæmia, and often there is very little evidence to the naked eye of a local reaction in the peritoneum. Free fluid and lymph may be practically entirely absent, but microscopical examinations show enormous numbers of meningococci present. Though involution forms are numerous, there are also present large numbers of what are evidently young active organisms. Where the dose has been very large, there may be practically no evidence of leucocytic emigration or endothelial proliferation; but where the dose is smaller, or where the animal has been recovering from the effects of the inoculation, the endothelial proliferation has been very marked, and the endothelial cells can be observed to be actively phagocytic towards the polymorphs, many of the latter being infected with the organisms, which may be seen inside the mononuclear cells in various stages of degeneration.

I have not been successful in experiments with guinea-pigs, even when young, and a goat inoculated subdurally (spinal) with a large dose of culture remained perfectly well. It is interesting to note the varying results which have been obtained by others in the case of goats. Heubner (31) succeeded in inducing a fatal meningitis, as did Councilman, Mallory, and Wright (32), but Bettencourt and Franca (33) failed; and though Albrecht and Ghon (20) had a fatal result, they failed to find the organism or indeed any pathological change in the brain or cord. It is probable that these varying results are to be explained by variation in virulence on the part of the organism, but it is possible that variation in the resistance of the animals has some influence. As has been said, the most important experimental work has been performed by Flexner (19) on monkeys. Bettencourt and Franca (21) had previously failed, but Von Lingelsheim and Leuchs (36) have also succeeded; and still more recently Flexner (37) has obtained still more striking results by direct introduction of the organism directly into the spinal subdural space. Flexner's results have been criticised on the grounds that enormous doses were used and that the disease was not perfectly reproduced. My own results will be published shortly, but I may briefly state that I have confirmed Flexner's

work, and by using fresh cerebro-spinal fluid containing the organisms, I have obtained a reproduction of the disease clinically and pathologically with smaller doses than Flexner used. I have also succeeded in producing a fatal toxæmia by introducing the organism into the sheath of the sciatic nerve, and in another case into the duodenum. In this latter case, the organisms were demonstrated in the mesenteric glands. There can be little doubt that further work in this direction affords the best hope for advance in our knowledge of the mode and paths of infection with the organism, and Flexner has already obtained results in the direction of combating the artificially produced disease in the monkey by the use of anti-sera.

The Relation of Epidemic Cerebro-Spinal Meningitis to Posterior Basal Meningitis.

Since Still's original description (22) of the bacteriology of this condition there has been considerable discussion as to the possible identity of posterior basal meningitis with the epidemic variety. As has already been said, Still indicated some differences between the behaviour in culture of the diplococcus he described and that of the organism originally described by Weichselbaum, and attributed considerable importance to the greater longevity of the organism (apart from the body), which he separated from a large number of cases. Undoubtedly, too, the symptoms of the two conditions do not quite correspond. Since his original paper, however, there has been a growing opinion that the differences are after all merely of degree, and can readily be explained on the hypothesis that in the epidemic form we are merely dealing with the same organism in a more virulent condition. All the evidence at our disposal shows that a variation in virulence is one of the marked characteristics of the meningococcus. Still showed that the arthritis and periarthritis sometimes seen in posterior basal meningitis were directly due to the same organism. We have already seen that this condition is occasionally met with in the acute epidemic form of the disease. It is true that cases of posterior basal meningitis have not, as a rule, the sudden acute onset which is so marked in the epidemic cases, and the duration of the disease in the former group is much more protracted. It is a common

observation that, roughly speaking, the total duration of the posterior basal meningitis cases runs to as many weeks as does that of the epidemic cases to days, and it could hardly be expected that the post-mortem appearances would be quite identical.

I may briefly refer to my own experience of this disease. In 1900 and 1901, during my tenure of a research scholarship of the British Medical Association, I undertook an investigation into the pathology of the condition. The want of time and space does not permit me to describe my results at any length, and the results of my research at that time were disappointing, largely owing to want of material. Since then, however, I have had a fairly large experience of the disease in my work at the Sick Children's Hospital, and I have altogether studied, from a pathological standpoint, over twenty typical cases. A detailed description of any of these cases would serve no useful purpose, as it would so largely be a repetition of what I have already described in the record of the acute cases.

I have been able to confirm to a very large extent Still's original work. A striking feature of the condition is the way in which the disease is so peculiarly limited to the central nervous system. I failed indeed to demonstrate the joint complications which Still so often met with. At the post-mortem on a typical case, one was struck with the absence of disease in other organs, and the comparatively small degree of general toxic change. A terminal broncho-pneumonia was occasionally seen, but apart from this there was little of moment to note. As regards the lesions in the brain and cord, the resemblance to the morbid anatomy of the epidemic cases was very striking, and the changes in many of the cases were absolutely indistinguishable from those seen in the more protracted of the acute cases. As regards the brain, though it is true that the condition is practically always more marked at the base of the brain, in many cases I have seen the purulent exudate spreading in the sulci, up to the extreme vortex. As one would naturally expect, in those more chronic forms of meningitis the after results of a resolving inflammatory condition are better seen. The inflammatory adhesions which ensue in the more severe cases very commonly lead to obliteration of the foramina of Majendie and Monro at the base of the brain, leading to a well-marked ac-

quired hydrocephalus. In those more chronic cases, too, small particles of inspissated lymph are frequently seen in the pia arachnoid, more particularly in the region of the posterior arachnoid cistern, where the purulent exudate in the acute stages so markedly tends to accumulate. One cannot help thinking that the appearance of a gelatinous exudate at the base of the brain, coupled with those small opacities, has led in the past, in some of those cases, to a naked eye diagnosis of tuberculous meningitis.

In the spinal cord the appearances, in a typical case of posterior basal meningitis, are also very characteristic. Indeed, one is left with the impression that the lesions here are often more marked, and of an older standing than those in the brain. All degrees, in a purulent leptomeningitis are met with. One sometimes sees the cord covered throughout its entire length with a thick, fairly uniform, gelatinous sheet of purulent exudate. The roots of the cauda equina are often found matted, due to a similar exudate. Sometimes the exudate on the cord is patchy in its distribution; in most cases the exudate is most marked posteriorly; and in resolving cases there may be practically no exudate left, though adhesions between the dura and pia arachnoid are apt to develop.

Into the histology of those cases I do not propose to enter, but I must refer to the bacteriology. The clinical diagnosis can be readily confirmed by lumbar puncture, and in practically all the cases I have studied, I have been able to demonstrate the very characteristic diplococci which are present in the polymorpho-nuclear leucocytes of the exudate. These diplococci have all the ordinary morphological characters of meningococci. In contrast to what one sees so often in the acute cases, free diplococci are comparatively seldom seen. Stained by Gram's method, the organisms I have found to be constantly Gram negative. As a rule, fewer organisms are found in the posterior basal cases, and in some cases a prolonged search is necessary to demonstrate the organism. The same, however, may be said about some of the acute cases, and even in a typical posterior basal case, the leucocytes are sometimes found crowded with diplococci. Involution forms of the organism have been, in my experience, much less numerous than in the typical acute cases.

I must now refer to the cultivation of the organism, and to

the results of animal experiments. In the great majority of the cases, I had no great difficulty in obtaining a culture from the cerebro-spinal fluid during life, for the first few weeks at least, in the history of the case. Naturally, as the organisms become fewer, it becomes more and more difficult to obtain a positive result by culture, but I have succeeded in a few cases, up to a period of five or six weeks during life; and even at the post-mortem, in much more protracted cases, I have obtained cultures from the purulent exudate in the lateral ventricles. The special point to note is, as Still has pointed out, that as all the organisms are not viable, it is necessary to smear large quantities of the exudate on the surface of the medium, in several tubes, to be sure of a successful result. As regards the media used, in many cases ordinary agar was all that was necessary, though blood agar was distinctly better. As regards the viability of the organism, my results do not quite correspond with Still's. As a general rule, it is true that the organism was more easily cultivated than the meningococcus of the epidemic cases, and grew in a much more vigorous manner, so that sub-cultures were not necessary every few days in order to keep the organism alive. At times, cultures were unsuccessful, where plenty of healthy-looking organisms were present in fluid; and even where a culture was obtained, on some occasions, the organisms, even on favourable media, died out very rapidly, so that it was impossible to keep the organism going without resort to very frequent sub-cultures.

As regards the naked-eye appearances, the grey-white or white opaque colonies described by Still were not constantly seen, and I have sometimes, in my cultures from the epidemic cases, seen colonies just as opaque as those derived from the posterior basal meningitis cases.

One of the reasons for the disappointing nature of the results of my research was my failure in my animal experiments. I used white mice, guinea-pigs, and rabbits. Intra-spinal inoculation in these smaller animals is unsatisfactory, owing to the limited sub-dural space, and the method I almost exclusively employed was intra-peritoneal injection. Still's results were fairly uniform and very satisfactory. I have come to the conclusion that the organisms from these cases which I have met with so far must have been much less virulent than those

described by Still. Had I employed the cerebro-spinal fluid itself for my injections I might have obtained better results. In a few cases, when large doses of the culture were given, the mice did die, and the diplococcus was demonstrated in films from the peritoneal exudate, and could be recovered in culture. Guinea-pigs, however, even when the animals were young, were almost immune, and rabbits practically entirely so. My efforts to exalt the virulence of the organism by passage through the peritoneal cavity of a series of mice also failed.

In spite of those differences from the organisms separated from the recent acute cases, I am left with the strong conviction that the organisms are practically identical. In posterior basal meningitis, it appears to me that we have to deal with a meningococcus where the vegetative phase in the character of the organism has been exalted at the expense of its virulence. As I have said, the more recent clinical observations, as for example those of Koplik (30), support the view that posterior basal meningitis is merely an attenuated form of epidemic cerebro-spinal meningitis. I may finally mention that though, as a rule, the cases of posterior basal meningitis I have seen have been characterised by the same irregularity in their distribution which has been noted so frequently in the case of the epidemic disease, there has been a slight tendency on certain occasions for the cases to occur in groups, and I have even known of more than one patient from the same household at the same time.

If the two conditions are really due to the same organism, we have a ready explanation of how the epidemic disease is kept alive, as it were. These cases of posterior basal meningitis may be regarded as so many sporadic cases of the disease. If this be true, it may be asked why, in a district where posterior basal meningitis is not so uncommon, at least in the spring months, we have so little experience of the epidemic variety. The recent severe winter may have had something to do with the extensive nature of the outbreak, but there are probably other general conditions in the resisting power of the general population, of which we know nothing as yet, which may act as essential predisposing factors. It is a reasonable assumption that, in such a time as the present, the epidemic cases should replace posterior basal meningitis ; and it is worthy of note that, in the opinion of

Dr John Thomson, only one of the meningococcal cases at the Children's Hospital this winter (Case No. 27) has quite conformed in type to the clinical picture of the more chronic disease as one has seen it in the past.

The Mode and Paths of Infection of the Meningococcus.

There seems little doubt that the meningococcus belongs to a group of organisms having their normal habitat about the nasal mucosa or upper respiratory passages, and there are now abundant observations on record of the organism having been demonstrated in this region. Undoubtedly many of the early reports are far from convincing, and as late as 1903 Lord (38) was only able to find three or four cases where the organism separated from the nasal mucosa corresponded in all particulars, cultural and otherwise, with true meningococci. Certainly observations made simply on stained films have little or no value. Among those nasal and throat bacteria are quite a variety of diplococci, one may say practically morphologically identical with the meningococcus. Many of them can be readily distinguished from the true organism by their cultural characters, but much more work must be done before the classification of this great flora can be considered at all complete. I do not consider it by any means proved that some of those diplococci may not yet prove to be merely attenuated meningococci, and it requires no effort of the imagination to foresee a time when so-called pseudo-meningococci may lead to much trouble in the way of bacteriological diagnosis. Gordon (28) has recently attempted a classification of the Gram-negative cocci found in normal saliva, but the basis of his classification is the power of those organisms to ferment a variety of sugars, and, as I have already stated, the place of this test in bacteriological diagnosis has yet to be firmly established. This fermentative power is, of course, not the only point on which Gordon founds his differentiation. Many of the diplococci grow at room temperature; others produce a definite colour in the colonies on artificial media; and one group gives rise to colonies showing marked coherency and a tendency to adhere to the medium. In spite of those fairly definite characters, it must be evident that the identification of the meningococcus in this situation,

even with the assistance of animal experiments, remains a matter of very considerable difficulty. The important point, however, for our present purpose is, that true meningococci have been demonstrated in the upper respiratory passages, not only of actually infected patients, but in the apparently healthy who have been in contact with the diseased. In the Silesia epidemic, Farrand (40) found the organism present in 23 per cent. of 635 cases examined. Jehle (41) has recently reported an investigation into the bacteria found in the nasal passages, etc., of miners in an infected district in Southern Germany, and endeavours to establish the theory that these adults may be important agents in transmitting the disease to their more susceptible children. His observations, however, have been already severely criticised by Krohne and Bahr (42 and 43).

From our study of the morbid anatomy of the condition, we have seen how often the base of the brain is the region where the infection is most marked, and it is a natural deduction that the ordinary mode of infection is by direct channels between the nose, etc., and the cranial cavity in the immediate vicinity. It must be admitted that such direct channels of communication do exist, for, as has been shown by Schwalbe and Kay and Retzius (44), the lymphatics of the nasal mucous membrane can be injected from the cranial cavity. This point, however, at once suggests a difficulty, at least in the case of patients suffering from the disease, for Flexner (19) has shown that, in the case of several monkeys given a spinal injection of the meningococcus, organisms morphologically identical with this organism were demonstrated in the nasal secretion of the infected animals. It is true that he failed to cultivate these organisms, so that absolute proof of their nature is as yet awanting, but the possibility that the organisms found in the nasal, and sometimes conjunctival, secretions may be due to a centrifugal spread, must not be lost sight of. If passage from the nasal mucosa to the base of the brain were the common mode of entrance of the organism, it might reasonably be expected that the lesions would be best marked at the anterior part of the base of the brain. The record of the post-mortem examinations, however, lends no support to this view. Allan and Dunn (46), in a carefully reported case of fulminating epidemic cerebro-spinal meningitis, occurring in the beginning of the Glasgow epidemic, examined the cribriform plate

and olfactory tracts. They found, however, that the olfactory epithelium gave no evidence of acute inflammatory change, and though some of the lymphatic spaces passing through the foramina of the cribriform plate showed leucocytic infiltration, it is evident, as they point out, that this is quite as likely due to a spread from within as a spread from without. As far as I have been able to find, there is no pathological evidence on record to show that the spread does take place from the nasal passages, and the same, in passing, may be said about the middle ear. It is plain, however, that there must exist some explanation for the marked basal character of the exudate, and it may be interesting and important to compare and contrast meningococcal with some other well-established varieties of meningitis. I am of opinion that a close analogy may be drawn between meningococcal and tuberculous meningitis. Let us look for a moment at the more important characters of the morbid anatomy in this latter disease. A common view, with regard to the etiology of this condition, is that the organisms reach the brain through the blood stream, and it need not be denied that, in some cases, this does occur. A marked character, however, of the exudate in tuberculous meningitis is that it is best seen posteriorly at the base of the brain, and this is certainly not what one would expect in a blood-borne disease. I have had a fairly extensive experience in tuberculous meningitis at the Sick Children's Hospital, and have been struck by the comparative rarity in children of the acute miliary tuberculosis of undoubted blood infection. In cases of general tuberculosis in children, tubercles are found in various organs, which can practically only be due to blood infection; but the host of extremely minute grey gelatinous tubercles which one is familiar with in the lungs, etc., of adults suffering from acute general tuberculosis is comparatively rare in the child. In the great majority of these cases, there can be little doubt that the lymphatic distribution of the disease is more important than the blood spread. Professor Greenfield has for many years taught that, in many cases of tuberculous meningitis, there can be shown in the spinal membranes older lesions than those seen in the brain, and he is of the opinion that tuberculous meningitis is not infrequently due to a lymphatic spread of the disease from the thorax or abdomen along

the nerve roots to the lymphatics of the cord, and thence to the base of the brain. This affords a ready and satisfactory explanation of the anatomical distribution of the basal exudate in this disease. It appears to me that some such spread is possible, if not indeed probable, in many cases of meningococcal infection. Granted that meningococci have found a suitable nidus for growth in the upper respiratory passages, in the buccal cavity, naso-pharynx, etc., various possible routes of entrance may be suggested. Thus, the tonsils and other adenoid tissue in the naso-pharynx may be really the channel of entrance. Just as in tubercle, the cervical glands and bronchial glands may become affected, and later the organisms may reach the lungs or pass along the lymphatics of the nerve roots to the surface of the cord. It may be pointed out that, in various epidemics on record, broncho-pneumonia has been frequently present in this disease, and in my own experience it has been very common. It may be reasonably urged, of course, that the pneumonia is secondary to a blood infection, or that it is due to associated pneumococcal infection, and we have positive information that such may occur. With regard to the infection of the cord, however, I am distinctly of opinion that the lesions here are often primary, at least as far as the central nervous system is concerned.

Another possible channel of invasion is open to the meningococcus. With an infective catarrhal process present in the upper respiratory passages, especially in the naso-pharynx, it can hardly be doubted that a considerable quantity of secretion containing the meningococci will be swallowed, especially by younger patients, and though the meningococcus is comparatively easily destroyed, it may, just as do cholera and typhoid bacilli, escape the normal gastric disinfection and reach the intestines, and entering by the lymphoid tissue, in the Peyer's patches, etc., reach the mesenteric glands, and later, by perineural lymphatics, the surface of the cord. Gastro-intestinal disturbance may possibly pave the way for such infection, and it is worthy of note that Lees and Barlow (47), in discussing the premonitory symptoms of posterior basal meningitis, describe such gastro-intestinal disturbance in quite a considerable proportion of their cases.

Gastro-intestinal changes have also been previously noted by several observers in acute meningococcal cases. Thus Radman (50) described changes in the mesenteric glands and intestines

which he considered were almost pathognomonic of the disease; and Cabra (51), in discussing the portals of entrance of the meningococcus, left open the question between a respiratory and a gastric or intestinal source of the infection. Symmers (52), in describing the morbid anatomy of some of the acute cases in the recent Belfast epidemic, drew attention to acute change in the intestines and mesenteric glands, though the changes he described evidently did not quite correspond to Radman's results.

We have further seen that, in the record of our own cases, slight degrees of gastro-enteritis have been not infrequently present, while in some cases an acute hæmorrhagic condition has been found, together with very distinct recent inflammatory condition of the mesenteric glands. One must, however, be cautious in regarding such changes as primary. They may, after all, be merely a secondary manifestation; in one of my experiments, where the organisms were introduced into the subdural (spinal) space in a monkey, a hæmorrhagic condition in the intestine was produced. It is notorious, also, that in quite a variety of acute infective diseases, in children especially, the lymphoid tissue in the alimentary tract may show marked secondary reactive change. The hypothesis of a primary intestinal infection hardly admits as yet of definite proof; but from a study of the mesenteric glands in such a case as that of Baby W. (No. 18), I am left with the impression that such infection is possible. A systematic study of the nerve-roots in very early cases may yield some definite information. There is practically constantly leucocytic infiltration in the perineural lymphatics near the cord, both in the natural and artificially produced disease; but the difficulty of distinguishing between a centrifugal and centripetal spread is always before one.

On examining the records of the Sick Children's Hospital during the past year, I have been struck with the number of cases of gastro-intestinal toxæmia. I shall content myself at present with briefly describing the main points in one such case.

John C., æt. 9 months, admitted July 28, 1906; died July 28, 1906. History.—For three days before admission, chokiness in throat and a cough; on admission, much collapsed, and cyanotic; hæmorrhagic discharge from the nose; lungs full of bronchitic râles; patient died a few hours after admission. Sectio, July 30.

Heart.—Showed cloudy swelling.

Lungs.—Congested, acute bronchitis, no pneumonia.
Liver, Kidneys, Spleen.—Show acute toxic change.
Intestines.—Show marked follicular catarrh.
Brain.—Not examined.

In other cases, the patients, mostly young children, have been taken acutely ill; some have shown hæmorrhagic eruptions; others laboured, irregular breathing, without definite pneumonic signs; while some have shown hæmorrhagic eruptions. The post-mortem appearances in those cases have been fairly constant; there has been evidence of a general acute toxæmia; the lungs have shown little beyond acute congestion, and, in some cases, early broncho-pneumonia. The intestines have shown marked follicular catarrh, and the brain merely undue hyperæmia of the meninges, without other gross lesion. I regret that in most of those cases I had no opportunity of making a bacteriological examination, and that they have not been sufficiently investigated. In their incomplete state they cannot be held to prove anything, but they are distinctly suggestive, and call for a complete bacteriological examination in acute gastro-intestinal toxæmias, occurring when meningitis is epidemic. These cases I have mentioned certainly do not correspond in type to the ordinary acute gastro-enteritis which is so frequent among children in the summer and autumn months. As regards the possible introduction of the organism in the food, *e.g.* milk, I know of no reliable data. One of my experiments indicates clearly that in the monkey at least an acute fatal toxæmia may be produced by introducing the meningococci into the duodenum.

In discussing the situation of the exudate in meningococcal meningitis, it is also interesting to compare the lesions with those seen in pyæmic and other cases where the blood-spread of the organism seems certain. In such cases the exudate is seen to be mainly vertical in its distribution. In the ordinary pneumococcal case this is also the rule.

We have positive proof that it is by no means uncommon for the meningococcus to reach the blood stream, and Andrews (48) has recorded a case where, in a rapidly fatal septicæmia, the meningococcus was cultivated from the blood, though, even at the post-mortem, microscopical examination failed to show the organism in the meninges. This, however, I expect, will prove to occur but rarely, and the appearance of the organism in the

blood is probably essentially a secondary change such as we see in so many infections, *e.g.* typhoid, where blood invasion was not so long ago thought to be very rare.

For a time I was under the impression that it was possible to found a naked-eye diagnosis of meningococcal meningitis on the peculiar tendency which the exudate on the sides and vertex of the brain shows in being limited to the sulci. A further experience, however, has convinced me that this is unsafe, and I believe that without a bacteriological examination it is impossible to distinguish with certainty between a meningococcal and a pneumococcal case. Though as a rule the exudate in pneumococcal meningitis is vertical, there seems no doubt that in certain cases it may be basal, but it may be fairly asked, is it certain that all pneumococcal cases are due to blood infection? May it not be that some are also due to lymphatic infection? It is not only possible, but probable that this does occur sometimes.

I have heard it argued that though the exudate in meningococcal cases is mainly basal, this may be secondary, and the same may be said about the more extensive exudate on the cord, the idea being that gravity associated with a looser arrangement of the pia-arachnoid in those situations may account for the greater quantity of exudate in those situations. The argument does not appeal to me. If it were correct, why should one so often see an appearance in a pneumococcal case such as in the patient C., Case No. 9, where, in spite of the extensive nature of the vertical exudate, the base of the brain and the cord are practically free. I do not deny that mere mechanical causes may play a part in the spread of a purulent exudate from the base of the brain to the cord, *e.g.* where you have a suppurative meningitis from middle ear disease extending along the spinal meninges, but I do not think that in meningococcal cases this is the correct explanation. It must be remembered also that there are other mechanical factors than gravity concerned in the spread of fluid in the cerebral sub-dural space at least, and free fluid (*e.g.* blood) in this situation may actually tend to rise towards the vertex.

Another argument one has heard used in explaining the greater amount of basal exudate is, that a developing hydrocephalus will exert more pressure on the vertex than at the

base, and limit the exudation in the former situation. In my experience I can only say that the degree of hydrocephalus necessary to cause such limitation only occurs late in the disease, and I have frequently seen much exudate at the base with very little at the vertex, though the convolutions give no evidence that there had been such marked intra-cranial tension. I am therefore left with the conviction that in the ordinary case some anatomical reason must account for the preponderance of the basal exudate, and the spread by the lymph stream appears by far the most satisfactory explanation.

As regards the general transmission of the disease, all the evidence at our disposal seems to indicate that direct transmission from one patient to another is distinctly uncommon. It would seem that some special susceptibility to the organism is necessary for infection, but it is more than likely that the apparently healthy may, through the presence of meningococci in their nasal and other secretions, act as transmittors of the disease.

If we regard posterior basal meningitis as identical with the epidemic form of the disease, it is evident that an attenuated form of the malady is practically constantly with us. From time to time it would appear that, for reasons still to be determined, the organism becomes more virulent and tends to produce, especially in children, a more acute infection, and that further, under those circumstances of exalted virulence the disease may become epidemic even among adults. In support of this contention one must remember that even among adults sporadic cases of the acute disease are seen from time to time. I investigated one such case while pathologist to the General Hospital in Birmingham in 1899. In this case typical meningococci were found. Some years ago I performed a post-mortem on another case, a patient of Dr Graham Brown's in the village of Blackhall, in the vicinity of Edinburgh. Though in this case I failed to find the organisms, the clinical history of the case, the post-mortem appearances, both naked eye and microscopic, and the absence of other organisms, left me fairly convinced that we had been dealing with an acute fulminant case of the disease.

What the conditions are which determine this increased virulence on the part of the organism have yet to be discovered. Climatic conditions, such as extreme degrees of cold, may aid,

though it may be noted in passing that some of the earliest fulminant cases in Edinburgh occurred in midsummer. I think it possible that when attacks of coryza are prevalent there is a tendency for pneumococci and other organisms practically normally present in the nasal and buccal secretions to become more virulent. The meningococcus may participate in this exaltation of virulence. Practically similar conclusions have been arrived at by Weichselbaum and Vansteenberghe and Grysez (49).

Suggestions as to Treatment based on the Pathology of the Disease.

In the first place, one cannot but be struck with the hopeless nature of the acute fulminant cases. The post-mortem appearances in the brain and cord indicate how futile it is to expect relief from the ordinary remedial measures, but, at the same time, the appearances in some of the more chronic cases indicate how much nature can do in the way of removing the inflammatory exudate. In the acute fulminant cases we have to deal with such a profound toxæmia that, short of serum therapy, which, as we shall see presently, cannot be said to show much promise at present, no drug administered in the ordinary way can be expected to prove of much benefit. The indications seem to be, in addition to treating the obvious symptoms, to dilute the toxin and aid in its removal as rapidly as possible. In such an apparently hopeless condition, the most heroic measures may be justifiable. For instance, it may be advisable to discuss the possibility of spinal lavage, and, under the circumstances, I see no reasons against a trial. A laminectomy might be performed high up in the spinal column, and the dura exposed. A fine cannula might then be introduced into the sub-dural space, and another low down in the lumbar region. A sterile, normal saline solution might then be allowed to slowly percolate between the two openings. It would be hopeless to expect that much, or even any, of the thick gelatinous exudate could be so removed, and, of course, much of the toxic products would remain untouched below the pia-arachnoid, but it appears that normal saline has a disintegrative action on the meningococcus itself, and some of the toxin might at least be diluted and removed.

Of course, I should not suggest such a line of treatment before its practicability and harmlessness had been first carefully tested on infected monkeys, but I certainly intend to attempt some such experiments.

The toxin in the general circulation might be diluted to some extent by copious intravenous transfusion of normal saline.

The injection of germicidal substances, such as collargol and lysol, as has been tried in several of the cases I have studied, does not seem to promise much, and the effect of such toxic agents on the central nervous tissue has yet to be determined.

As regards the possibilities in the way of serum treatment of the disease, Flexner's results may be considered at least more hopeful, but there is a widespread belief among pathologists of the general inefficacy and even danger in the use of anti-sera in such a disease as this. As will have been noted, several of the patients whose cases I have discussed have been treated by injections of serum; both Kolle's and Burroughs, Welcome & Co.'s anti-sera having been administered. I am not convinced that they have not done actual harm. Though congestion of the internal organs is quite a feature in the untreated disease, I am of opinion that the acute congestion of the lungs has been even more marked in some of the patients treated with the serum.

More hope is attached to the possible use of a vaccine as a prophylactic agent, but as there is apparently comparatively little risk of direct infection from patient to patient, it is questionable whether such prophylactic injections would be justifiable.

The only other suggestion I have to make is one which has been already advanced by many authorities, viz. the systematic lavage with mild antiseptic solutions of the nose and naso-pharynx of those in contact with patients suffering from the disease.

Conclusions.

The following deductions seem permissible from what has been found in this investigation:—

1. That the recent epidemic in Edinburgh and district has been definitely due to the meningococcus.

2. That in the course of an epidemic of meningococcal menin-

gitis, pneumococcal cases may occur which can only be recognised by a bacteriological examination.

3. That while the meningococcus can in the great majority of cases be obtained in pure culture from the cerebro-spinal fluid during life in the earlier stages of the disease, at a later period other organisms may invade the brain and cord, and that consequently post-mortem bacteriological results are not so reliable and possibly account for some of the confusion which has arisen as to the etiology of the disease.

4. That while the organism in the great majority of cases practically conforms to the original description given by Weichselbaum, variation occurs in the staining properties of the meningococcus, and that in particular the statement that the organism gives a constant and definite negative reaction to Gram's stain is not justified.

5. That the statement by Gordon, in his recent report to the Local Government Board, "that the meningococcus never forms even short chains" is also erroneous.

6. That the organism grows but feebly apart from the body, even in the most favourable artificial media, and that the chances of its continued growth and development under natural conditions apart from the body are extremely slight.

7. That there exists a marked variation in the virulence of the organism separated from the living tissues, as tested by experimental inoculation in the lower animals.

8. That direct transmission of the disease from patient to patient is rare.

9. That posterior basal meningitis is pathologically identical with acute cerebro-spinal meningitis, the differences between the clinical features of the two conditions being due to a varying virulence on the part of the organism.

10. That the existence of posterior basal meningitis and cases of sporadic acute cerebro-spinal meningitis prove that the disease is more or less constantly present with us, and that epidemics of the acute disease are accounted for by conditions still to be determined, which lead to exaltation of the virulence of the organism or a diminished resistance on the part of a certain section of the general population.

11. That though the disease is only exceptionally infectious from patient to patient, when an epidemic is prevalent there is

a widespread distribution of the organism, possibly through the agency of slight coryzas in patients not obviously the subjects of serious acute disease.

12. That in such times of epidemic the small percentage of the population, mostly infants, susceptible to the milder form of the disease—viz. posterior basal meningitis—shows a heightened susceptibility, leading to an acquiring of the acute form of the disease; and further that, possibly from the wider dissemination of the organism under such circumstances, the disease may spread to the more susceptible among the adults.

13. That in the ordinary case the organism, having found a lodgment in the upper respiratory passages, reaches the central nervous system by lymphatic channels, and that a case has been made out for a fuller investigation into the possibility of the channels of entrance being the lymphatics of the neck, thorax, or abdomen, communicating with the corresponding spaces around the nerve roots of the spinal cord.

14. That at a later stage the organisms may be disseminated by the blood stream, but that the essential symptoms of the disease are mainly produced locally in the central nervous system.

15. That pneumonic complications are not uncommon, and that a gastro-intestinal type also occurs. Whether the gastro-intestinal lesions are primary or secondary must still be left an open question.

16. That in addition to the obvious lesions produced in the meninges, grave histological changes occur in the substance of the brain and cord and in other internal organs from the general toxæmia which is present.

17. That a marked reactive change on the part of various endothelial cells, designed for a phagocytic purpose, is a histological feature of the disease.

18. That the disease can be reproduced in its clinical and pathological entirety by intra-spinal injection of the organism with its products in monkeys, and that a favourable field is thereby afforded for a fuller investigation of the pathology and treatment of the disease.

19. That as all the indications are that the essential toxine belongs to the endo-cellular variety, serum therapy is not yet a practical possibility.

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Abstracts

ANATOMY.

ON THE ORIGIN OF THE VAGUS FIBRES TO THE LUNG.

(339) **(Ueber den Ursprung des Lungenvagus.)** IKEGAMI and YAGITA, *Okayama-Igakkwai-Zasshi*, March 31, 1907.

THE writers had come to the conclusion, from previous research, that the unstriped muscle of the lungs receives its motor fibres not from the vagus but from the sympathetic, and the present research was conducted to determine this point.

Their research was carried out on dogs and their method was: (1) division of the vagus above the plexus nodosus; (2) division of the vagus below the plexus nodosus; (3) extirpation of the base lobe of the right lung. The dogs were killed three or four weeks after the operation, and the nerves examined by Marchi's method, the cells of the plexus nodosus by that of Nissl.

Their conclusions are (1) that the motor fibres of the vagus do not supply the lung, because the extirpation of the base-lobe produces no particular change in the vagal nuclei, and the cutting of the vagus above the plexus nodosus causes no degeneration of the nerve fibres on the bronchi; (2) that the changes in the plexus nodosus and the degeneration of fibres in the lung show that the vagus branches consist of sensory fibres which originate from the plexus nodosus; (3) the cells of origin lie especially in the central part of the plexus nodosus, and make up about one-eighth of the cells of the plexus. JOHN D. COMRIE.

PHYSIOLOGY.

STIMULATION OF THE CEREBELLUM. (Über Reizungen des (340) Kleinhirns.) A. LOWRIE, *Neurolog. Centralbl.*, 16 Juli, S. 642.

LOWRIE first gives a short account of previous experimental researches on this subject, omitting, however, reference to those of Russell and Horsley. He then describes briefly a series of personal investigations, carried out chiefly on dogs. At first he had been puzzled to find that stimulation of one inferior gyrus semilunaris caused in some cases curving of the spine, sometimes to the same and sometimes to the opposite side, in other cases only to the opposite side. The explanation of this was found to be as follows: When the electrode was applied too much to the side, irritation of the dura took place. When this was carefully avoided the results were constant, and curving of the spine always occurred with the concavity to the contra-lateral side. On the other hand, the muscles of the neck and limbs always reacted only on the stimulated side.

All the experiments gave results that pointed strongly to the conclusion that no localising centres exist in the cerebellum. Stimulation of a gyrus semilunaris inferior gave identical results with stimulation of the corresponding side of the vermis.

A special series of experiments was performed with the view to confirming Ferrier's teaching as to the eye movements that occur in relation to stimulation of different parts of the cerebellum. No correspondence with Ferrier's views on this matter was obtained; several times movement of one eye alone took place on stimulation of the gyrus. ERNEST JONES.

PATHOLOGY.

THE SPINAL LESIONS OF HERPES ZOSTER. (Les lésions
(341) **medullaires du Zona.)** ANDRÉ-THOMAS et LAMINIÈRE, *Revue Neurologique*, July 30, 1907, p. 693.

A CAREFUL study of two recent cases of uniradicular herpes zoster.

1. The short fibres for the grey matter of the posterior horn come to an end, for the most part, at the level of their entry into the cord. Some pass up to higher segments, in one case from the ninth dorsal to the sixth, and in the other from the eighth dorsal to the fourth.

2. The fibres to the column of Clarke enter in the same segment as the root to which they belong. They may be traced also to the segment just above.

3. In one case it was possible to follow the long fibres of the eighth dorsal root to the nucleus of the column of Goll; they spread out dorsally and ventrally as they ascend.

4. The zone of Tissauer is not affected by degeneration of the roots.

5. Descending radicular fibres are easily traceable; in the case of the eighth dorsal lesion they descend three segments.

6. Most interesting is the presence of vascular congestion, hæmorrhagic foci, and disturbance of the grey matter, all on the side of the herpes and in the region of the lateral horn. The association of the intermedio-lateral tract (some of the cells of which are affected) with the sympathetic suggests an involvement of that system in the disease. It is quite possible that the cutaneous phenomena are due, not so much to the irritation or destruction of the ganglion or the root, as to the simultaneous alteration of the sympathetic fibres which join the radicular fibres, and are comprised in the same inflammatory focus. S. A. K. WILSON.

ON CHANGES IN THE GANGLION CELLS OF THE SPINAL
(342) **CORD IN EPIDEMIC CEREBRO-SPINAL MENINGITIS. (Ueber Veränderungen der ganglienzellen des Rückenmarks bei der Meningitis Cerebrospinalis epidemica.)** LUDWIG, *Deut. Zeitschr. f. Nervenheilk.*, Bd. 32, H. 4-6.

THIS paper offers an explanation of the marked muscular atrophy which is often noticeable in the course of this disease.

The writer examined sections of the spinal cord, peripheral nerves, and muscles in eight fatal cases of acute meningitis, and his findings are, generally speaking, as follows. The muscles very

frequently showed a great multiplication of nuclei, atrophy of many of the fibres, unequal staining reactions of different fibres, and, in various places, loss of transverse striation. The nerves showed some degenerated fibres, some with swollen axis cylinders, and frequently a honeycomb appearance from changes in the sheaths. The cord showed sometimes small hæmorrhages, and from its anterior horn-cells the Nissl's bodies had usually been reduced to small granules, or had totally disappeared; the nuclei were frequently quite structureless in appearance and sometimes vacuolated, and there was frequently pigmentation of the cell bodies.

The writer discusses the pathological bearings of these changes, and suggests a relationship between cerebro-spinal meningitis and poliomyelitis. JOHN D. COMRIE.

CULTIVATION OF THE MENINGOCOCCUS IN THE PRESENT (343) EPIDEMIC OF CEREBRO-SPINAL MENINGITIS IN BELFAST. SYMMERS and WILSON, *Brit. Med. Journ.*, June 22, 1907.

THE writers record the result of examining the fluid obtained by lumbar puncture in thirty-one cases of epidemic cerebro-spinal meningitis. From twenty-seven of these the *diplococcus meningitidis* was obtained by culture. In its identification the characters upon which reliance was placed were: (1) The colonies upon a mixture of peptone agar and ascitic fluid are smooth circular discs, with regular contour and of translucent grey colour; (2) the colonies consist of diplococci, with numerous isolated cocci; (3) the organism is gram-negative; (4) it does not grow at 20°-22° C.; (5) it produces acid in media containing dextrose and maltose, but not in those containing galactose and saccharose.

They also obtained the meningococcus from the blood in three out of fifteen cases of the disease. JOHN D. COMRIE.

ON THE DIFFERENTIATION OF THE MENINGOCOCCUS FROM (344) OTHER GRAM-NEGATIVE DIPLOCOCCI IN THE NASO-PHARYNX OF CEREBRO-SPINAL FEVER CONTACTS. BUCHANAN, *Lancet*, June 8, 1907.

As the result of prolonged investigation in the public health laboratory of Glasgow, the writer agrees with Gordon as to the importance for identification of the meningococcus of the following points: the power to produce an acid reaction in bouillon containing sugars, which is not effected by the micrococcus catarrhalis;

and the temperature test, the meningococcus failing to grow at 23°-25° C. As an improvement upon the reaction test, he uses a solid medium, consisting of ox serum (3 parts), bouillon (1 part), glucose (1 per cent.), and neutral-red (1 in 10,000). Upon this medium the meningococcus shows a distinct pink reaction in 18 hours. JOHN D. COMRIE.

THE OPSONIC INDEX AND AGGLUTINATION IN CEREBRO-
(345) **SPINAL MENINGITIS.** ALICE TAYLOR, *Lancet*, July 6, 1907.

THE writer records a series of careful observations upon eight cases of cerebro-spinal meningitis, in which she determined the opsonic index for Weichselbaum's diplococcus, at the same time noting the power of the serum of these patients to produce clumping of the organisms.

Her conclusions were that a high index to the meningococcus is present in all cases of the disease, though it varies greatly in height according to the age of the culture used. The index to other organisms, *e.g.* the tubercle bacillus, is unaffected. A high index, if obtained, is diagnostic of the disease, though with certain strains of the organism a low index does not necessarily exclude it. The clumping took place during the incubation of serum and diplococci (ten minutes), and was observed in the same slide on which the organisms were counted for the opsonic index. JOHN D. COMRIE.

A CASE OF CEREBRO-SPINAL MENINGITIS; ISOLATION OF
(346) **THE SPECIFIC ORGANISM; PREPARATION OF A VACCINE; RECOVERY.** RUNDLE, MOTHAM, WILLIAMS, ORR, and WILLIAMS, *Lancet*, July 27, 1907, p. 220.

THE patient was a boy of six months; his condition was such as to justify a most unfavourable prognosis when vaccine treatment was commenced about a month after the onset of the disease. The opsonic index was at first low. Five inoculations in all were made; after each the index rose, on three occasions very materially. Apyrexia followed each injection, and there was a steady amelioration of the symptoms—vomiting, head retraction, squint, Kernig's sign, hyperæsthesia, and emaciation progressively disappearing in the course of a month.

In this case the agglutination reaction was present, but was not strictly specific. J. HARVEY PIRIE.

MACROSCOPIC AND MICROSCOPIC ANATOMY OF EPILEPSY.

(347) **(Anatomie macroscopique et microscopique de l'epilepsie.)**
D. DE BUCK, *La névraxe*, Vol. ix., 1907.

THE writer, who believes that idiopathic epilepsy is a neuro-autocytoxic disease, has examined the central nervous system of twenty epileptics to ascertain whether the histological appearances are in favour of this view. The tissues were fixed either in 10 per cent. formal, 3 per cent. bichromate of potass, or Flemming's chromo-osmic fluid, and were stained after the methods of Nissl, van Gieson, Heidenhain (iron hæmatoxylin), Pal, Flemming or Cajal (for neurofibrils). Macroscopically the organs did not show any specific lesion, the cerebro-spinal fluid was not increased; pronounced atrophy and sclerosis of the cornua ammonis was only met with three times, and he attaches no special importance to this sign in epilepsy.

He does not specify from what regions his cortical sections were taken. The microscopical changes found in the nerve cells were characteristic of a chronic regressive atrophic type; in only one case were they of a hypertrophic type. The earliest lesion, in less advanced cases, was diffuse chromatolysis, passing progressively up to complete achromatosis. Frequently there was a granular and finely vacuolated condition of the cytoplasm, and some cells showed fissures around the nucleus. Finally, there was atrophy and progressive molecular disintegration. The dendrites were the first to succumb, then the cell body. The cytoplasmic disintegration was preceded by a fatty degeneration. The neuro-fibrils were more resistent than the chromatoplasm; they progressively disappeared, however, from the centre of the cell body towards its periphery and the dendrites, and in advanced stages no trace of fibrils remained. The nucleus became dense, homogeneous, irregular and atrophied, contemporaneously with the cytoplasmic changes, but was not swollen as in *reaction á distance*. He does not agree with Clark and Prout that the cortical cell lesion is primarily of nuclear origin. Lesions of the nerve cells in the cerebellum, basal ganglia, pons and cords were of the same nature, but rarely passed the first stages of chromatolysis. He noticed in sections treated by Pal's method, a rarefaction extending even to an entire disappearance of medullated fibres, beginning in the tangential zone, and encroaching progressively to the supra- and intra-radiary, and even extending to the radiary fibres, thus confirming the observations of Claus and Vanderstricht, Alzheimer, Rosenfeld, Weber, etc., and he also observed the nodular state, the beaded appearance of the fibres, which precede their disappearance, and which he looks upon as pathological, and comparable to that

observed in the fibres of the cord and nerves under toxic influence. He rarely met with the increase and thickening of the glia on the surface of the molecular layer, the sclerotic hypertrophy, which, according to some, plays such an important rôle in the genesis of epilepsy, and the cases in which it did occur were usually associated with idiocy. He noticed a fairly constant relation between, on the one hand, the gravity and rapid progress of the epilepsy, and, on the other hand, the activity and proliferation of the neuroglia in the cortex. He was not able to assure himself, however, that the glia cells (or the blood cells) played a neuronophagic rôle, nor was he able to detect fatty degeneration of the glia cells. Variable and inconstant lesions were noted in the vessel walls, dilatations, and small peri-adventitial hæmorrhages, which he considers are secondary phenomena, and masses of large fatty granules.

The course of both cell and neuroglia lesions is from the surface to the depths of the cortex, so that the small pyramidal cells are more affected than either the large pyramidal or polymorphic cells.

He concludes that the appearances are in favour of the cyto-toxic nature of epilepsy, and whilst he cannot at present affirm that there is a histological picture pathognomonic of the disease, thinks that there are legitimate hopes of finding such in the future.

The pathological features which he regards as most peculiar to epilepsy are the progress of the cortical lesions from without inwards, and the finely vacuolated state in the chromatolytic cells. This vacuolated condition he has found in all his cases, whilst he has vainly sought for it among the brains of dementia precox, confusional insanity, and general paralysis.

JOHN TURNER.

PATHOLOGICAL ANATOMY AND PATHOGENESIS OF ACUTE
(348) **ENCEPHALITIS. (Anatomie pathologique et pathogénie de l'encéphalite aigue.)** LAIGUEL-LAVASTINE AND VOISIN, *Arch. de méd. expérimentale*, January 1907, p. 28.

ACUTE encephalitis may in certain instances be considered as a morbid entity, determined in the same way as other organic affections due to general toxæmia.

Anatomically, acute encephalitis may be diapedetic, purulent, or hæmorrhagic in character. In the first case, one meets with all stages, from discrete leucocytic infiltration to abscess formation. The purulent variety is the latest stage of the preceding, and is distinguished by its mechanism from cerebral abscess of embolic origin.

The hæmorrhagic type consists in inflammatory reaction of the parenchyma and sometimes of the meninges, with parenchymatous hæmorrhagic foci from phlebitic or arterial lesions.

Pathogenetically, no distinction can be drawn between diapedetic encephalitis and analogous processes in other viscera. Purulent encephalitis is the last stage in inflammatory reaction.

Hæmorrhagic encephalitis results either because of the virulence of the infection producing congestive rather than diapedetic phenomena, or because of a predisposing fragility on the part of the blood vessels of the brain. S. A. K. WILSON.

THE CEREBRO-SPINAL FLUID IN HEREDITARY SYPHILIS.

(349) **(Le liquide céphalo-rachidien des hérédo-syphilitiques.)** RAVAUT, *Ann. de derm et de syph.*, February 1907, p. 81; RAVAUT et DARRÉ, *Gaz. des Hôp.*, February 12, 1907, p. 207.

RAVAUT'S paper is based on the study of twenty-eight cases of hereditary syphilis, the ages of which ranged from a few days to one year. Out of eight cases in which there were no lesions of the skin or mucous membranes, spinal lymphocytosis was absent or slight in seven, and moderate in one. Out of 20 cases presenting lesions of the skin or mucosæ, in four the reaction was slight or absent, but in these cases only the buttocks were affected; in ten there was a moderate reaction. Examination of the fluid for the spirochœta pallida was always negative, as was also the inoculation of monkeys with the fluid, both before and after centrifugalisation. Ravaut thinks that in hereditary as in acquired syphilis, spinal lymphocytosis has the same value as a lesion of the skin or mucosæ, both from the standpoint of diagnosis and as a guide to treatment. It must not be supposed that every nervous symptom found in heredo-syphilis must necessarily be associated with a reaction of cerebro-spinal fluid. Many such symptoms are due to banal causes not connected with syphilis, such as gastro-intestinal disturbance or rickets. The true significance of such symptoms is determined by the character of the cerebro-spinal fluid. It must also be remembered that in the young child numerous affections, *e.g.* pneumonia, gastro-enteritis, otitis, and mumps (v. *Rev. of Neurol.*, April 1907, p. 301), may be accompanied by a meningeal reaction. But the lymphocytosis found in these affections is transitory, and soon disappears after the subsidence of the acute disease, whereas the reaction determined by syphilis is much more tenacious, and may persist for months. An instructive case is recorded by Ravaut and Darré in a girl, aged three months, suffering from hereditary syphilis, in whom they found a meningeal reaction lasting five and a half months, of

which the different phases could be followed by comparative study of the clinical symptoms and the results of lumbar puncture. At five different times convulsive attacks occurred, which on each occasion were less intense in their manifestations and duration. At the end of five and a half months they disappeared completely, and never returned, though the case was followed till the child was three years and four months old. Lumbar puncture showed that the lymphocytosis, which was very intense at first, diminished little by little, and disappeared completely simultaneously with the clinical symptoms. It is interesting to note that there was no increase of the lymphocytosis at the moment of the convulsive attacks. The lymphocytosis in this case was due to syphilis, because the patient had no other affection associated with analogous meningeal reactions, *e.g.* disturbance of the digestive or respiratory systems or mumps. Further, its definite disappearance under the influence of treatment was in favour of syphilis. This case illustrates the diagnostic and therapeutical value in hereditary syphilis of a persistent lymphocytosis of the cerebro-spinal fluid, accompanied by intermittent meningeal phenomena. During the intervals of the attacks, the absence of any clinical phenomena might have made one presuppose an integrity of the nervous system, whereas lumbar puncture indicated its impairment. So, while the lymphocytosis persisted, energetic treatment was employed by injections of grey oil and mercurial inunction.

J. D. ROLLESTON.

PITUITARY INSUFFICIENCY IN INFECTIOUS DISEASE. (Sur (350) un syndrome d'insuffisance hypophysaire au cours des maladies toxi-infectieuses.) AZAM, *Thèse de Paris*, 1907.

THIS study is based on observations made in Renon's service at La Pitié. Pituitary insufficiency is characterised by two principal symptoms, which are a fall of the arterial tension and an acceleration of the pulse. Among secondary symptoms are noted insomnia, loss of appetite, frequent sweating, and disagreeable sensations of heat. The administration of powdered pituitary substance in doses of thirty or forty centigrammes daily was followed in all cases by a rise of arterial tension, slowing of the pulse, and an increase in its force and amplitude. In some cases there also occurred an increase of diuresis and a rise in weight, as well as a disappearance of the secondary symptoms of pituitary incompetence. Owing to its effect on the arterial tension pituitary substance is contra-indicated in two conditions: tuberculosis complicated by hæmoptysis, and typhoid complicated by intestinal

hæmorrhage. Among the patients subjected to this treatment were cases of typhoid, pneumonia, influenza, puerperal fever, laryngeal diphtheria, and cerebro-spinal meningitis.

J. D. ROLLESTON.

CLINICAL NEUROLOGY.

MULTIPLE UNILATERAL PARALYSIS OF CRANIAL NERVES.
(351) **(Paralysie unilatérale de nerfs craniens multiples.)** LEJONNE et OFFERT, *Rev. neurologique*, July 15, 1907, p. 637.

DURING an attack of enteritis the patient was suddenly attacked by paralysis, involving the whole of the left facial nerve in particular, but also affecting the motor fifth, the hypoglossal, the olfactory, upper trigeminal, and glosso-pharyngeal on the same side. Under the influence of a simple electric treatment nearly all the symptoms have disappeared. The diagnostic problem consists in the differentiation of a local basal meningitis from a polyneuritis. In this connection the complete conservation of ocular movements is of some significance. Probably the lesion was a non-specific localised polyneuritis. Reference might here be made to an instructive paper by v. Fränkl-Hochwart on analogous cases.

S. A. K. WILSON.

A CASE OF ACUTE POLIOMYELITIS. BARNES and MILLER, (352) *Brain*, June 1907, p. 101.

A CASE OF ACUTE POLIOMYELITIS IN AN ADULT, WITH (353) **MARKED BULBAR AND OCULAR SYMPTOMS. MICROSCOPICAL REPORT.** PERKINS and DUDGEON, *Brain*, June 1907, p. 110.

A CASE OF RELAPSING ACUTE POLIOMYELITIS, SHOWING (354) **NYSTAGMUS; WITH PATHOLOGICAL EXAMINATION.** REGINALD MILLER, *Brain*, June 1907, p. 117.

OF these three cases the chief interest of the first two lies in the pathogenesis of the condition. The first case was that of a boy of 9 who died eight days after the onset of the illness. Paralysis was almost complete up to the neck, but apparently there was no disturbance of sensation. The pia-arachnoid was found to be congested; the lower cervical cord was soft, swollen, and showed punctate hæmorrhages. Other parts showed similar but less marked appearances. Cultures of *staphylococcus pyogenes citreus*

were obtained from the cord. Microscopically the vessels in the anterior fissure were dilated, and there was marked perivascular infiltration. This condition was more pronounced where they entered the white matter; the lateral and posterior arteriole as well as the central were affected.

The changes in the white matter were very slight, but in the grey matter there were hæmorrhages, chiefly in the anterior cornua, but also in the posterior and general cellular infiltration. The cells taking part in this change fall into two groups: (1) large numbers of cells indistinguishable from lymphocytes; (2) larger cells with large pale-staining nuclei and a relatively large proportion of non-granular protoplasm.

The latter the authors consider probably originate from the adventitia of the vessels (but they might be proliferated neuroglial elements), and appear to have no relation with polymorpho-nuclear leucocytes.

The nerve cells were in various stages of degeneration. There was no evidence of thromboses in the vessels entering the cord. Micro-organisms were not found in any of the sections.

The second case was that of a man of 26, who died seven days after the onset of the disease. His symptoms were mainly bulbar —difficulty in swallowing, etc; before death there was also diplopia and diaphragmatic paralysis. Bacteriological examination of the blood was negative; further investigations were not made. In the bulb and cord was found a great perivascular infiltration, chiefly by lymphocytes, and there was also a marked neuroglial proliferation, but the authors did not think any of the cells originated from the adventitia. The spinal meninges showed a similar infiltration, and the vessels were much engorged with red blood corpuscles. There was no thrombosis. In the white matter there was considerable fatty degeneration near the periphery. The mode of onset—sore throat, faucial hyperæmia, intense congestion of trachea and bronchi, and engorgement of lungs—suggests an infection primarily in the fauces.

The third case was in a boy of 3½, who lived for seventeen weeks after the onset. The initial attack was severe, and he had three relapses, the first two indicating further involvement of the cord, while the third consisted in the sudden onset of violent nystagmus, which lasted for twelve days. The autopsy was not made till six days after death. The vessels of the cord were greatly congested, particularly in the grey matter, but also in the meninges. There were no thromboses. The perivascular infiltration consisted chiefly of lymphocytes, with others which the author thinks were plasma cells. Patches of the grey matter were similarly infiltrated, and some areas were necrotic with proliferating neuroglia cells surrounding them.

There was also inflammatory foci in the medulla, pons, and cerebellum; many nerve cells were totally destroyed, while near by might be others almost normal. The destruction was only within the areas of cellular infiltration, which would suggest that the nerve-cell degeneration was due immediately to the inflammatory process, and not to any toxin.

The case would seem to support the primarily inflammatory rather than the primarily vascular theory.

J. H. HARVEY PIRIE.

(355) **SENSORY AND MOTOR DISTURBANCES IN PARTS ABOVE THE DISTRIBUTION INVOLVED BY DEFINITE ORGANIC LESIONS OF THE SPINAL CORD.** WEISENBURG, *Journ. Nerv. Ment. Dis.*, July 1907, p. 434.

TWO cases are described in which there occurred, secondary to a transverse myelitis, a progressive diminution or loss of cutaneous sensibility above the level of the original lesion, extending for several segments. This progressive change was noted in each instance a considerable number of years after the accident. In addition, increased reflectivity in the upper extremities was remarked. The author attributes this apparent change in sensation (unfortunately the examinations were made by different observers) to "progressive arterial obliteration and necrosis in the contiguous parts." His statement that "these facts have not, heretofore, been *noted*," is scarcely accurate. S. A. K. WILSON.

(356) **ON A CASE OF SPASMODIC PAINFUL SYRINGOMYELIA OF RAPID DEVELOPMENT. (Sur un Cas de Syringomyélie Spasmodique Douloureuse a Évolution Rapide.)** HENRI VERGER, *L'Encéphale*, July 1907.

A WOMAN, aged 50 years, who had suffered from pains in the arms for many years, noticed that her fingers were becoming flexed into the palms, and that her forearms were wasting. The arms were held stiffly from the side, the forearms flexed, the wrists extended, and the fingers flexed into the palms. The muscles of the forearms were atrophied, and the thenar and hypothenar eminences flattened. The reactions both to faradism and galvanism were diminished, but there was no reaction of degeneration. The left arm was more markedly affected than the right. On the outside of the left forearm there was an area of anæsthenia to temperature and pain, with intact sensibility to

touch. There was no scoliosis. There was some weakness of the legs, with increase of tendon reflexes and no atrophy. At night the patient experienced severe shooting pains in both arms and both legs. In six months the arms were completely helpless, and the legs were in a state of spastic paraplegia, which made walking impossible. The progress of the case was extremely rapid, death occurring twenty-one months after the first appearance of symptoms. The cord was found to be flattened transversely, enlarged, soft, and pale from the level of the second cervical roots down to that of the seventh cervical. On section there appeared a central cavity throughout the region of the cervical enlargement, and a smaller cavity, with harder walls, in the right half of the dorsal region of the cord. There was no pachymeningitis. Microscopically the grey matter in the cervical region was found to be totally destroyed by a central glioma, with cavity formation, and round about this glioma there was a diffuse sclerosis, involving chiefly the lateral columns. The nerve roots were also affected by the sclerosis. In the dorsal region the gliomatous process was entirely limited to the grey matter on the right side, and ceased at the lower end of the dorsal cord. Sclerosis of the pyramidal tracts could be traced right down to the sacral division of the cord.

HENRY DUNBAR.

THE LARYNGEAL SYMPTOMS IN MULTIPLE SCLEROSIS.

(357) (**Die laryngealen Erscheinungen bei multipler Sklerose des Gehirns und Rückenmarks.**) L. RÉTHI. Wien. Verlag von JOSEF ŠAFÁŘ. 1907.

DR RÉTHI gives in this monograph a detailed description of the appearances to be found in the larynx in disseminated sclerosis, and discusses their possible explanations. He begins with the consideration of cases previously published: the first (Leube) dates from 1871. In addition details are given of 10 cases observed by himself. He has collected in this way altogether 44 cases in which the larynx has been examined; this includes two cases in which a *post-mortem* examination was made.

The greater part of the work is devoted to a discussion of the semeiology. The slurring speech Réthi considers to be due to a disturbance in the breathing—a failure to supply promptly sufficient pressure of air for phonation. The monotonous voice, the sudden changes in pitch and breaking into falsetto, arise from lack of control of the intrinsic muscles of the larynx, the last symptom from a weakness of the thyro-arytenoidens externus muscle.

The laryngeal paralyses are not true paralyses in most cases, but are rather due to weakness, as Gottstein has shown, unless the bulbar nuclei are themselves affected, in which case the corresponding paralysis is seen on examining the larynx. Some authors point out the rarity of paralysis, but emphasise the numerous conditions simulating it; others, again, state that paralysis frequently occurs. In Réthi's collection, in 15 cases out of 44, some form of adductor paralysis was present, but the condition never remained constant in any one of these cases; in many of them the tensors of the cords were also affected. Adductor paralysis was present in 13 cases—in six paresis, in seven paralysis—thus, in 63 per cent. of his cases some form of paralysis was present.

Tremor, one of the most important symptoms in this disease, must necessarily be mainly lateral in direction, when occurring in the vocal cords. It may, however, also manifest itself as an alternate lengthening and shortening of the cords. This symptom is observed in about one half of all cases. Lateral tremor may occur on phonation or on forced inspiration. The tremor may also be seen occasionally in the extrinsic muscles, especially the sterno-thyroid and thyro-hyoid muscles. Tremor may be observed in a large number of other diseases, including hysteria, chorea, paralysis agitans, tabes, syringomyelia, bulbar paralysis, and syphilis.

The question is next discussed as to the nature of the tremor: is it the result of irritation, or due to a disturbance of co-ordination, or merely an evidence of weakness? Réthi favours the last view.

The sensory disturbances in the larynx are finally shortly referred to. Throughout the book, the work of other observers is abundantly quoted and discussed. An index of authors is added, so that reference is easy. W. G. PORTER.

(358) **MENINGISM.** FRANK E. TYLECOTE, *Med. Chron.*, June 1907.

TRANSIENT, but nevertheless definite, symptoms of meningeal irritation are not uncommon in the specific fevers, and are often described as pseudo- or threatened meningitis. In a few cases the duration of these symptoms is longer, and their intensity greater, and yet, whenever such a case comes to autopsy, no naked-eye meningeal changes will be seen. Two groups of cases may be recognised: in the first, or organic group, there is probably a very minor degree of inflammation—an extension from neighbouring structures, as in those cases in which the symptoms ensue in the course of complicating otitis media; in the second, or functional group, there is no such inflammatory focus, and the condition is evidently a toxic one. Meningism is then tentatively defined as a

functional condition caused by the selective action on the meninges and cerebral cortex of the toxins circulating in the blood-stream in these diseases. It occurs in scarlet fever, diphtheria, measles, mumps, influenza, typhoid, and in tubercular subjects, in whom it often lasts some time, and leads to a diagnosis of tubercular meningitis being made, which is not borne out by the after-history of the case. Lumbar puncture is most valuable in making a differential diagnosis between meningism and meningitis, and in some cases relieves the symptoms. The most constant sign met with in meningism is retraction of the head, with which opisthotonos is often associated. The presence or absence of pyrexia does not seem to influence the symptoms. The child usually lies on the side, with legs drawn up; he is usually extremely irritable, and may whine incessantly. The following summary of the points, helping one in differentiating meningism from meningitis on the clinical symptoms, will indicate the other main points in the symptomatology: (1) In the acute infections meningism is often early in its occurrence and of short duration; this does not hold good of the more chronic conditions such as tuberculosis. (2) Kernig's sign is, as a rule, absent in meningism. (3) Pyrexia is more often absent in meningism than in meningitis. (4) The coincident presence of opisthotonos is in favour of meningism. (5) Lumbar puncture is more often beneficial in meningism than in meningitis. (6) Frequent recurrence of the symptoms following lumbar puncture favours an organic lesion. (7) Marked involvement of cranial nerves favours meningitis. (8) The onset of the symptoms in question, for the first time during the defervescent or convalescent stage of an acute infection, is much against meningism. (9) The onset of meningism is acute, while in meningitis the symptoms more often develop gradually. (10) The symptoms once developed, usually remain of the same intensity till they begin to decline—there is not the ebb and flow, or the gradual increase in intensity, often seen in meningitis. (11) Hyperæsthesia and tâche cérébrale are more frequent in meningitis. (12) Absence of marked progressive wasting, during the period when the symptoms are present, favours meningism. (13) In fevers the absence of a predisposant to meningeal infection, such as trauma or otitis, or of family history of tubercle or its presence in the patient, favour meningism. (14) Slowing of the pulse and irregular respiration, so common in meningitis, are rare in meningism. The literature relating to the subject is somewhat fully referred to and a bibliography given, the points to be noted as regards lumbar puncture and the examination of the fluid are given in detail, and short abstracts are given of seven illustrative cases.

AUTHOR'S ABSTRACT.

MENINGOCOCCUS SEPTICÆMIA. SIMON, *Journ. Am. Med. Assoc.*, (359) June 8, 1907, p. 1938.

THE writer records a case of cerebro-spinal meningitis in which the meningococcus of Weichselbaum was obtained from the blood during life, and which he believes to be only the eighth case so far recorded in which this organism was so obtained.

There was a leucocytosis of about 50,000, with polymorphonuclears, 62 per cent.; large mononuclears, 21 per cent.; and small mononuclears, 17 per cent.; no eosinophiles or mast cells among 500 cells counted. About 8 per cent. of the leucocytes contained diplococci, and the large mononuclears appeared to be as actively phagocytic as the polymorphonuclears. In one c.mm. of blood there were 7380 diplococci.

References are given to the cases already placed on record.

JOHN D. COMRIE.

CEREBRAL HYPERÆMIA AS A FACTOR IN THE THERA-
(360) **PEUTIC ACTION OF LUMBAR PUNCTURE.** FRANK C. EVE, *Lancet*, July 27, 1907, p. 228.

THE author considers that the chief factor in the beneficial action sometimes seen following on lumbar puncture in cases where the pressure of the cerebro-spinal fluid is *not* raised is the passive hyperæmia of the cerebro-spinal vessels which must ensue. His remarks are illustrated by a case of tetany which resisted six weeks of various forms of treatment, but improved rapidly and completely after the removal of an ounce of cerebro-spinal fluid. The pressure at time of removal was 200 m.m. of H_2O.

J. H. HARVEY PIRIE.

CONTRIBUTION TO THE KNOWLEDGE OF CIRCUMSCRIBED
(361) **ADHESIVE CEREBRAL ARACHNITIS. (Zum Kenntnis der umschriebenen Arachnitis adhaesiva cerebralis.)** PLACZEK and KRAUSE, *Berl. kl. Wochenschr.*, July 22, 1907.

THE writers record in detail a case which presented the following symptoms: Vomiting, headache, vertigo, enfeeblement of gait, tendency to fall to the left, almost complete external ophthalmoplegia of both eyes, complete right-sided facial paralysis. Ophthalmoscopic examination showed no abnormality. A lengthy discussion of the differential diagnosis is given, with the grounds upon which was founded the diagnosis of a tumour in the posterior cranial fossa.

An operation, of which the details are given, was undertaken, but no tumour was found. There was, however, widespread adhesion between the upper surface of the cerebellum and the tentorium, with great collection of cerebro-spinal fluid in the space thus shut off. Nevertheless the patient began at once to mend. The facial paralysis passed off, the power to move the eyes returned; and although the temperature fluctuated considerably, at the end of eight weeks it became normal, and the power of all parts of the body had returned completely.

The condition is compared by the writers to the meningitis serosa spinalis described by Oppenheim and others.

JOHN D. COMRIE.

(362) **CASE OF CEREBRAL TUMOUR WITH ABOLITION OF THE TENDON REFLEXES. (Cas de Tumeur Cérébrale avec abolition des reflexes tendineux.)** A. VAN GEHUCHTEN, *Le Névraxe*, Vol. ix., 1907, p. 39.

OF the various fibres whose terminal ramifications are in connection with the anterior cornual cells, Van Gehuchten maintains that the cortico-spinal are the only ones whose function is inhibitory. All the rest have a stimulating action on the anterior cornual cells, maintaining the normal muscular tonus. If the cortico-spinal fibres be interrupted, reflex muscular tonus is increased. If any of the other fibres (rubro-spinal, vestibulo-spinal, posterior longitudinal bundle, reticulo-spinal, and the collateral reflex fibres of the posterior roots) be damaged, the result is a diminution or loss of muscular tonus. The state of the deep reflexes is independent of modifications in the muscular tonus, since we may have flaccid or hypotonic muscles together with exaggeration of the deep reflexes, as, for example, in certain cases of flaccid hemiplegia, etc.

Van Gehuchten divides cutaneous reflexes into two groups: firstly, an upper or cortical group of reflexes, which require integrity, not only of the reflex arc, but also of the cortico-spinal fibres, and secondly, a lower or medullary group of reflexes belonging to the spinal cord proper, and best seen in cases where the cord is liberated from all influences coming from the higher centres. There is, in certain cases, a contrast between the deep reflexes and the cortical group of skin reflexes. Thus, in many cases of hemiplegia and paraplegia, we have exaggeration of the deep reflexes with loss of the "upper" cutaneous reflexes. The deep reflexes are not cortical in origin, inasmuch as they are not abolished but exaggerated in lesions of the cortico-spinal path. Nor are they entirely spinal in origin, since in total transverse

lesions of the end they are abolished. The latter fact is explained by assuming the loss of some influence on the anterior cornual cells by descending fibres other than the cortico-spinal ones—whether vestibulo-spinal, reticulo-spinal, or rubro-spinal.

Van Gehuchten relates a case of cerebral tumour in a man of 39, in which, during the last three weeks of the patient's life, there was total loss of knee-jerks with persistence of the skin reflexes, plantar, cremasteric, and abdominal. The plantar reflexes were flexor in type. The deep jerks in the upper limbs were also lost during the last few days. Autopsy showed a tumour of the lenticular nucleus and island of Reil, which had compressed and driven toward the right the mesencephalon and adjacent parts of the floor of the third ventricle. Sections stained by the Marchi method showed no degeneration of the motor path, nor of the rubro-spinal, vestibulo-spinal, or reticulo-spinal paths. There was slight diffuse degeneration in the posterior columns of the cord.

Raymond (*Archives de Neurologie*, p. 1, 1904) also published a similar case of cerebral tumour, in which the deep reflexes were abolished and the plantar reflexes were normal, but there is no note of the autopsy. Raymond explains the loss of the deep reflexes in his case as due to compression of the cord, producing the well-known degeneration of the posterior roots which is so often demonstrable by the Marchi method. Van Gehuchten, on the other hand, attributes the disappearance of the deep reflexes to loss of function of the rubro-spinal fibres with integrity of the cortico-spinal paths, the skin reflexes being therefore preserved. He maintains that degeneration of the posterior columns of the cord cannot account for loss of the deep reflexes, since such loss of reflexes is rare, whereas posterior root degeneration is common in cerebral tumours. Moreover, it is difficult to explain on a posterior root theory why only the fibres for the deep reflexes should be thrown out of action and not the fibres for the skin reflexes. Nor can such a theory account for the absence of sensory changes or of ataxy, such as accompany the loss of deep and skin reflexes in tabes. Van Gehuchten, therefore, considers the simplest and most natural explanation of the loss of deep reflexes in such cases to be the loss of function of the rubro-spinal fibres, the escape of the skin reflexes being attributed to the integrity of the cortico-spinal path. PURVES STEWART.

(363) THE CEREBRO-SPINAL FLUID IN PARESIS, WITH ESPECIAL REFERENCE TO ITS CYTOLOGY. WILLIAM BURGESS CORNELL, *Am. Journ. of Insanity*, July 1907, p. 73.

IT was in 1901 that lumbar puncture first came into general use as a recognised procedure in diagnosis. Referring to the advances

made in the technique of cerebro-spinal cytodiagnosis, the author insists on the superiority of the method devised by Fuchs and Rosenthal in 1904. The centrifuge is dispensed with, and the cells in a definite volume of undiluted fluid are counted. For this purpose a special cell-chamber of known capacity as made by Zeiss is employed.

The author arrives at the following conclusions:—

1. Every case of paresis exhibits a spinal leucocytosis and increase of albumen.

2. This sign is also, from point of view of its constancy, in all probability the earliest.

3. The diagnostic value of a negative puncture is often of greater value than a positive one.

4. The cell-counting method with Fuchs and Rosenthal's slide is more accurate and rapid than the centrifuge technique, and has the great advantage in permitting comparative results.

5. The use of Onna's polychrome blue in the *mélangeur* permits a simultaneous differential count.

6. A differential count is important in differentiating the paretic fluid from others, especially where the cytosis is due to a small number of polynuclears.

7. The conditions under which syphilis produces a spinal leucocytosis demand further investigation, especially regarding the number and character of the cells. The increase of cells in the paretic fluid is apparently independent of any long antecedent syphilis.

8. There seems to be a correlation, both analitative and enertitative, between the spinal and hæmic leucocytosis, which particularly refers to the mononuclears, but includes the polymorphonuclears, especially after convulsions.

EDWIN BRAMWELL.

A STUDY OF REFLEXES OF THE LOWER EXTREMITIES
(364) **IN SIXTY CASES OF PARESIS, WITH A SPECIAL REFERENCE TO THE PARADOXICAL REFLEX.** GORDON, *Journ. Nerv. Ment. Dis.*, July 1907, p. 430.

IN 60 cases of general paralysis the paradoxical "reflex" was present in 42 instances; in 35 cases on both sides, and in 7 on one side. The "old" triad of reflex phenomena, viz. exaggerated knee-jerk, ankle clonus, and Babinski's sign, is here markedly dissociated. While the first was present in the majority of the 60 cases, the other two were rare. Oppenheim's reflex also was infrequent in the series. In view of the fact that the pyramidal paths are frequently involved in the disease, in a majority of cases

only to a slight extent, the existence of the paradoxical phenomena is, according to the author, of considerable clinical significance.

S. A. K. WILSON.

TUBERCULIN THERAPY IN GENERAL PARALYSIS. (**Zur Tuber-**
(365) **kulintherapie bei progressiven Paralyse.**) B. PILCZ, *Wien. Med. Woch.*, 20 Juli, S. 1462.

THIS article is largely a restatement of Pilcz's published work (*Jahrb. f. Psychiatr.*, Bd. xxv.) on Von Wagner's treatment of G.P., 1. This is based on the fact that temporary improvement often follows intercurrent pyrexia in the disease: $\frac{1}{100}$ of a mgm. of the old tuberculin is injected, and the dose rapidly increased. Four cases are shortly reported in which brief improvement was observed. The writer recommends that the treatment be given a trial in early cases, such as are seen by practitioners.

ERNEST JONES.

VAGAL AND VASO-VAGAL ATTACKS. Sir WILLIAM GOWERS,
(366) *The Lancet*, June 8, 1907.

THE term "vagal attacks" is used as a designation for prolonged seizures, the symptoms of which consist chiefly in disturbance of some of the functions of the pneumogastric, for the most part sensory and subjective. With the vagal symptoms there is often combined a slight mental change and also disturbance of the vaso-motor centre causing constriction of the vessels, and coldness, specially of the extremities. Associated with the latter may be some sensory impairment, and sometimes also a form of slight tetanoid spasm. These features vary much in relative proportion.

The vagal symptoms are chiefly sensations referred to the stomach, the respiratory system, and the heart. At least we may probably ascribe to the gastric nerves a sensation referred to the epigastrium, described as a sense of oppression or of fulness, but often indescribable. It begins suddenly, irrespective of the state of the stomach or of its functions, and often seems to ascend to the chest, very seldom to the throat or head, as does the aura of epilepsy. There is seldom nausea and never vomiting. Even more common, especially as an early symptom, is a sense of respiratory distress, of difficulty in breathing. It is sometimes so intense as to amount to orthopnœa and to compel the sufferer, if lying, to sit upright, although there is no corresponding sign of impairment of breathing. With this may be combined cardiac symptoms, discomfort, acute pain in some cases, often a sensation of sudden stoppage of the heart, followed by rapid action, With

the dyspnœa, or the cardiac sensation, or both, is often associated a sense of impending death, so intense that no recollection of its falsity in preceding attacks prevent the conviction of its present reality. It naturally causes alarm, but apart from this there is sometimes a sense of intense fear and dread, which is recognised to have no adequate cause.

Although there is no impairment of consciousness in typical cases, a peculiar slight mental state is common, and may even be the first symptom. It is generally described as a difficulty or slowness of mental operations, a difficulty in thinking or in concentrating attention. Trifling as it may seem, it always begins suddenly and strikes the patient as a state quite unlike the normal condition. Sometimes it involves a slowness in speaking, but this seems partly due to the sense of dyspnœa. Perhaps allied to this is a sudden sense of physical fatigue, which also is often an initial symptom.

The vaso-motor spasm sometimes attains a high degree. To it the symmetrical coldness is certainly due, for the pulse becomes small at the same time. When general there is pallor of the face. Shivering is common and may amount to a definite rigor, but this occurs when the coldness is beginning to lessen. With the coldness of the extremities tingling and numbness in them are often described, and sometimes there is the tetanoid spasm we shall consider later.

A fairly typical case was a single woman, aged 28 years, whose cousin was epileptic. She had suffered for nine months from attacks that were heralded by occipital headache on waking in the morning. Sometime in the forenoon she suddenly found that she could not fix her attention on any subject. After this had lasted an hour or so a sense of sleepiness came on; then suddenly she felt wide awake with an intense sense of fear, extreme coldness of the hands, feet, and legs, and inability to move. Presently a peculiar, indescribable sensation was felt at the epigastrium and back, sometimes with slight nausea. After a few minutes of this, violent beating of the heart set in, accompanied almost at once by an intense sense of suffocation. If lying she had to sit up to breathe, though there was no evidence of dyspnœa. Then the attack subsided, as a flush developed, attended by slight perspiration and a flow of tears (without any sense of emotion). The flush was accompanied by a sense of warmth, beginning in the head and passing to the arms, but with this there was always a slight distinct rigor. The teeth would chatter and the hands would shake. It was greater the more severe the attack. This soon ceased and she was well. During the attack there was a curious sense of unreality in all she saw and afterwards objects were blurred and indistinct. Some more severe attacks began with tingling

in the hands and arms, face and trunk; the epigastric sensation developed to definite nausea; the general coldness was extreme, "as if made of stone," and the violent action of the heart was preceded by a feeling that it had stopped and that she was dying.

Cases are also met with which seem to establish the position of the attack in the borderland of epilepsy. They may be regarded as cases of *petit mal* extended, protracted, drawn out, as it were, so that the initial sensory stage, instead of being momentary, often continues for a long time. It may even subside without the usual loss of consciousness. S. A. K. WILSON.

ON THE OCCURRENCE OF EPILEPTOID ATTACKS IN TACHY-
(367) **CARDIA AND BRADYCARDIA.** F. H. CLARKE, *Brit. Med. Journ.*, Aug. 10, 1907, p. 308.

THE clinical features of two cases of tachycardia and one of bradycardia, in which there were epileptoid attacks, are described by the author. In the first case, at varying intervals, the heart's action became very rapid and irregular. When the irregularity had continued for some time, pauses were noticeable in the radial pulse; and if such a pause lasted from six to nine seconds, the face became pale, the lips livid, there was a scream, the thumbs were drawn inwards, the head backwards, and there was convergent strabismus. In the more severe attacks, consciousness was lost. The severity of each attack was proportional to the length of time the radial pulse was absent. At any stage of the fit, a return of the pulse cut short the attack. In the second case, the attacks of paroxysmal tachycardia were accompanied by fits, precisely similar to epileptic seizures. In the third case, the man's pulse rate is usually 40 per minute. But it often falls below this, and at such times, if the patient exert himself unduly, either physically or mentally, he is apt to become suddenly pale, and to lose all sense of his surroundings for a few moments. W. T. RITCHIE.

CRISES OF ANGUISH WITH IMPOSSIBILITY OF MAINTAIN-
(368) **ING THE SITTING POSITION, ETC. (Crises d'anxiété avec impossibilité de garder la position assise ou de rester immobile dans la position verticale, persistant depuis plus de cinq ans chez un jeune garçon.)** X. FRANCOTTE, *Journ. de Neurolog.*, 20 juillet, p. 261.

THE gist of this interesting case is as follows. A boy of nine, after being terrified by his schoolmaster, became a prey to crises during which he could not endure either to sit or to stand, but had the

irresistible need to walk. The crises lasted half an hour or longer. They occurred daily at first, but later only every two or three months. They were accompanied by intense fear of falling. The doctor to whom he was taken first threatened to thrash him, and then held him down on a table during a crises.

Francotte discusses the relation of the above psychasthesic manifestation to that described by Haskovec under the name of *Akatisie*. It differed from this, however, in the two matters, that sitting in itself did not necessarily bring on an attack, and was well borne apart from the attacks; whilst, on the other hand, the attacks frequently occurred when the boy was up.

ERNEST JONES.

A CASE OF HYSTERICAL RETENTION OF URINE WITH LONG
(369) **DURATION. (Ein Fall von dauernder hysterischer "Retentio urinae.")** J. RAIMIST, *Neurolog. Centralbl.*, 16 Juli, S. 646.

THIS condition is usually of short duration, though cases are on record in which catheterisation had to be performed for several weeks. The case here recorded is therefore of unusual interest.

In April 1903 a Jewish boy, then aged twelve, escaped the Kischinen butcheries by hiding, with thirteen other Jews, in casks in a cellar, where they were in imminent peril. For three days he had neither food nor drink, and during this time did not evacuate either urine or fæces. A week later pain and tiredness in the legs set in, and soon developed into paraplegia. This lasted for two years and four months (August 1905). In August 1904 retention of urine began, and was complete from the start. He was in the hospital for four months, being cathetised daily, but left in the same condition. No further change occurred until he entered Raimist's clinic, June 1906. He had thus passed urine for nearly two years, only with the aid of a catheter. This he had used himself several times a day, and it was striking that there was no evidence of vesical infection in spite of the fact that no aseptic precautions whatever were taken, and the instrument was kept in his trouser pocket.

On examination a number of signs of hysteria were found, the most noteworthy being a contraction of the visual field of an amazing extent. No attempt was made to cure the hysteria, but the symptom of retention was removed by suggestion, so that after a few days, during which urethral pain was marked, he could urinate freely.

ERNEST JONES.

UNILATERAL PARALYSIS AGITANS OCCURRING AFTER
(370) **HEMIPLEGIA.** SAILER, *Journ. Nerv. Ment. Dis.*, July 1907, p. 425.

THIS is the brief record of a case of a woman who, at the age of 69, developed right hemiplegia, from which she made a partial recovery. Four years later she first noticed tremor in the arm and hand of the opposite side, which has continued until the present time. There is now the typical symptom-complex of paralysis agitans in the left arm, with paresis of the left arm and tremors in both feet.

The remarks on the determination of the localisation of the lesion are quite incomplete. No reference is made to the association of disease involving the rubro-spinal tracts with tremor: the statement is made that "the majority of neurologists now believe that the lesion is situated in the cerebral cortex" (*sic*). The author makes no allusion in text or bibliography to the work of Gordon Holmes on the relation of tremor to certain lesions of the mid-brain, and the assumption is apparently made that a "Parkinsonian hemiplegia" is identical with the disease known as "paralysis agitans." S. A. K. WILSON.

POST-HEMIPLEGIC HEMI-TONOCLONIC SYNDROME. (**Syn-**
(371) **drome Hémi-tonoclonique Post-hémiplégique.**) G. ÉTIENNE, *L'Encéphale*, July 1907.

THE case recorded is that of a woman who, seven years ago at the age of 49, had a complete right hemiplegia, evidently due to a cerebral hæmorrhage, and associated with impairment of sensibility, both to touch and pain over the whole of the right side. The muscular contraction of the right side which ensued was not of the common post-hemiplegic order. The muscles were in a state of active tonic contraction, accompanied by a great deal of pain; and the slightest peripheral stimulation brought on an almost intolerable exacerbation of this myotonic condition, to which would often be added paroxysms of violent clonus, lasting usually for about a minute. These attacks almost always followed automatic involuntary movements of the limbs, and were hardly ever brought on by deliberate voluntary movements. The muscles of the right side, particularly of the right arm, were markedly hypertrophied, and this was due to a true increase of muscular tissue, not to œdema, or to any pseudo-hypertrophic process. The patient's condition has gradually become worse, until at the present time she is practically helpless by reason of the very painful paroxysms brought on by any attempt at walking.

The writer discusses fully the pathological significance of this syndrome, and concludes that in the present case the lesion involves practically the whole of the optic thalamus, with pressure on the internal capsule and descending sclerosis of the pyramidal tract. HENRY DUNBAR.

INVESTIGATIONS ON THE PUPILS IN THE EPILEPTIC, THE (372) HYSTERICAL, AND THE PSYCHOPATHIC. (Pupillenuntersuchungen an Epileptischen, Hysterischen und Psychopathischen.) MAX LINDE, *Psychologische Arbeiten*, herausg. von E. KRAEPELIN (of Munich), Bd. v., H. 2.

THE examination included the determination of the diameter of the pupil, the extent and time of the pupillary reaction, the nature of the psychical reflex. To elicit the latter, a sudden sound was used as a stimulus, pain was produced by a variety of stimuli, various olfactory stimuli were employed, but discontinued, as the form of stimulus involved too many factors. The instrument used to determine the size of the pupil was that of Weiler. The following were the steps in each examination:—

1. Illumination of both eyes with feeble light; measuring of both pupils. 2. Illumination of the right eye with bright light, of the left with feeble light; measuring of the right pupil. 3. Illumination of the left eye with bright light, of the right with feeble light; measuring of the left pupil. 4. Illumination of both eyes with bright light; measuring of both pupils (secondary reaction of Weiler). 5. Illumination of the left eye with bright light, of the right with feeble light; measuring of the right pupil (consensual reaction). 6. Illumination of the right eye with bright light, of the left with feeble light; measuring of the left pupil (consensual reaction). 7. Determination of the psychical reflex. 8. Estimation of the reaction time of the pupillary light reflex.

Seventy cases were examined, with the following results:—

1. The epileptic, hysterical, and psychopathic show the same pupillary characteristics.

2. The pupil is on the average wider than in the normal individual.

3. Inequality of the pupils was found in 27 per cent.

4. The pupillary reaction-time varied greatly, but on the average was longer than in the normal individual.

5. The consensual reaction was in some cases less extensive than the direct reaction.

6. The secondary reaction of Weiler was always present.

7. The reaction to psychical stimuli was always present.

C. MACFIE CAMPBELL.

ON THE PROGNOSTIC SIGNIFICANCE OF THE ARGYLL-ROBERTSON PHENOMENON. (Zur prognostischen Bedeutung des Argyll-Robertson'schen Phänomens.) L. W. WEBER (of Göttingen), *Monatsschr. f. Psych. u. Neur.*, March 1907. (373)

THE author makes the following additions to the communication of Pilcz on the above subject (*vid.* abst. *Rev. of Neur. and Psych.*, March 1907, p. 225). A moderate dose of alcohol, as Cramer has shown, may cause in imbeciles, degenerates, and exhausted individuals various pupillary anomalies, among which is failure of reaction to light (? with intact accommodation). Weber has frequently seen in cases of cerebral arteriosclerosis complete absence of reaction to light (? with intact accommodation), sometimes of a recurrent nature. (Bumke is very cautious as to the occurrence of true Argyll-Robertson pupils in arteriosclerotic and senile dementia.) C. MACFIE CAMPBELL.

ON CLONIC CONTRACTIONS IN GENERAL PARALYSIS CONSIDERED SYNCHRONOUS WITH THE PULSE. (Ueber die sogenannten rhythmischen, mit dem Puls synchronen Muskelzuckungen bei der progressiven Paralyse.) O. FISCHER (of Prague), *Monatsschr. f. Psych. u. Neur.*, March 1907. (374)

IN 1905 Kemmler called attention to the fact that in general paralysis there occurred attacks in which the clonic contractions appear synchronous with the pulse. Fischer confirms the observation, but has studied the phenomenon more accurately. Even without the use of apparatus, one notices that the synchronicity is not exact. In four cases the author took tracings of the muscle contractions and of the pulse, and found that there was no true synchronicity; the number of muscle contractions was, as a rule, not very different from the pulse-rate. In other cases where the same exact method was not followed, he could also determine the absence of synchronicity. C. MACFIE CAMPBELL.

ÜBER DIE AUSFALLSERSCHEINUNGEN NACH LÄSIONEN DES CENTRALNERVENSYSTEMS. MAX ROTHMANN, *Neurolog. Centralbl.*, 1 Juli, S. 594. (375)

THIS article consists of a theoretical discussion chiefly on the subject of the Ersatz and Diaschisis hypotheses. Mainly as a result of his experimental researches, Rothmann finds Von Marakow's

"action at a distance" view unsatisfactory as regards the explanation of asphasia, the motor consequences of cerebral lesions, etc. He puts forward a hypothesis of his own, which, shortly stated, is that the distant effects of local lesions, particularly of the cortex, are greater in man than in apes, and greater in apes than in dogs, because the higher the animal in the evolutionary series, the more do the phylogenetically ancient centres and paths become subordinated to the higher centres, and so less capable of independent action. ERNEST JONES.

MOTOR APHASIA AND ITS CORTICAL LOCALISATION.

(376) **(L'aphasie motrice et sa localisation corticale.)** DÉJÉRINE, *L'Encéphale*, May 25, 1907, pp. 471-520.

THE author describes in great detail the clinical observations and pathological findings in two cases of motor aphasia. Pathologically, the lesion involved the cortical and subcortical matter of the posterior end of the third left frontal convolution, as well as of the adjacent part of the second frontal convolution: further, it extended to the cortex of the anterior marginal fissure and to the adjacent digitation of the anterior convolution of the visual (in one case); part of the corona radiata of the frontal lobe and the "faisceau compacte" of the corpus callosum were also implicated. Evidently the distribution of the lesion corresponded to certain vascular areas. Considerable stress is laid on the fact that in neither case was the operculum injured, which shows that the centre for movements of the lower part of the face, the tongue, the pharynx and larynx, is outside Broca's zone. It is important to note, in view of Professor Marie's contention, that the posterior convolution of the insula, the three posterior digitations of the anterior convolution of the insula, the external capsule, Wernicke's region, the retro-insular area, the basal ganglia and the internal capsule, were all intact.

The two cases show that motor or Broca's aphasia may be produced by a limited lesion in the anterior part of the speech area, that is, in Broca's area, without implication of the operculum, the motor convolutions, the temporal lobe, or the basal nuclei.

S. A. K. WILSON.

MUSICAL AMNESIA IN A CASE OF SENSORY APHASIA.

(377) **(Amnésie musicale chez un aphasique sensoriel, ancien professeur du musique. Conservation de l'exécution, de la lecture, de l'improvisation et de la composition.)** LAMY, *Rev. neurologique*, July 30, 1907, p. 688.

THE subject of this paper is an old man at present living in the Hospice of Bicêtre, who was formerly a professional musician, and

who spends his time still in giving music lessons to the children in the place. He has suffered for four years from incomplete sensory aphasia, presenting both jargonaphasia and jargonagraphia. His appreciation of what is said to him is fairly good; he can read and copy. His spontaneous speech, however, is frequently unintelligible, intercalated as it is with meaningless words, and the same is true of his handwriting.

The minute examination of the condition of his musical faculty can scarcely be summarised satisfactorily.

He hears and understands musical sounds; he plays the violin, writes and copies music; he composes and improvises in a very correct and pleasing manner. On the other hand, his musical memory is absolutely nil. He does not recognise the air which he has just improvised; he does not recognise his own compositions, nor such hackneyed pieces as "Au clair de la lune" or "J'ai du bon tabac."

It is obvious that a cursory examination might determine a diagnosis of "musical deafness" corresponding to "word deafness" in this patient, for he does not recognise the Marseillaise when he hears it. But what has been said of the case shows that he is not afflicted with amusia, but with musical amnesia. In this connection it is interesting to note that the defect of memory applies, not to recent acquisitions, but to popular airs and songs of his childhood, which is contrary to the general law of disintegration of memory. The contrast between this defect and the actual condition of the patient's musical technique is remarkable.

There seems to be a conservation of what is automatic, mechanism, rhythm, the comprehension and use of conventional signs, and this is coupled with a loss of the musical auditory memory. S. A. K. WILSON.

PSYCHIATRY.

TRANSITORY ALCOHOLIC PSYCHOSES. (Transitorische Alkohol-
(378) **psychosen.)** F. CHOTZEN (of Breslau), *Monatsschr. f. Psych. u. Neur.*, April 1907.

THE various transitory mental disorders due to the reaction of psychopathic individuals to alcohol are difficult to analyse, in view of their close relationship to transitory hysterical and epileptic psychoses and to similar psychoses in non-alcoholic individuals of psychopathic constitution. In the characteristic psychopathic intoxication (pathologischer Rausch) the symptoms last longer than the excitement and unrest due to the ordinary physiological

intoxication; the picture presented may be that of a hysterical or epileptic complex, with additional features due to acute alcoholic intoxication.

In addition to this characteristic psychopathic intoxication, there are observed crepuscular states (Dämmerzustände) of simple nature or complicated with a rich variety of delusions; a third group is formed by cases closely allied to the psychopathic intoxication, but of longer duration and symptomatologically more complex. It is this third group of transitory alcoholic psychoses which the author discusses in relation to six cases which he reports. The most prominent features of such psychoses may be delirious, motor, expansive, or depressive symptoms. The first four cases cleared up in a few days. The fifth case reported presented a psychosis of several weeks' duration, with a crepuscular state forming merely one episode in the course of the whole psychosis; such a case is probably of degenerative origin. The sixth case, an alcoholic, forty-two years of age, presented periodic depressive attacks. The relation of such attacks to manic-depressive insanity, and the exact rôle played by the alcohol, are discussed.

C. MACFIE CAMPBELL.

(379) **ON THE RELATIONS BETWEEN REACTION ERRORS AND SOUND ASSOCIATIONS. (Ueber die Beziehungen zwischen Fehlreaktionen und Klangassoziationen.)** R. HAHN, *Psych. Arb*, herausg. v. E. Kraepelin (of Munich), Bd. v., H. 2.

THE aim of this experimental investigation was to determine the relationship between certain abnormal phenomena in states of psychomotor excitement. In such states the reaction-time is lowered, according to previous investigators, and errors are more frequent; sound associations are more common than in the normal condition. Do these phenomena always go together? To produce the psychomotor excitement various agents were used—alcohol, walks, cycling, abstention from sleep, hunger, learning by heart. The effect of alcohol was rather to increase the reaction-time; the increased facility of discharge of the reaction being more than balanced by the increased difficulty of grasp of the stimulus. The number of errors in reaction (reacting with the wrong hand to the vowel given as stimulus) was as a rule little different from the normal. Sound associations and rhymes were more frequent under the influence of alcohol; the grasp of stimuli was much impaired by the poison. Previous investigators considered that the decrease in the average reaction-time, under the influences mentioned, took place at the expense of the accuracy of the reactions; but in the author's experiments the accuracy of the reactions showed no

marked deviation from the normal. As a matter of fact, the mistaken reactions only exceptionally coincided with a short reaction-time; there was no question, therefore, of short reaction-times being due to precipitate responses without choice of the nature of the response. The results of the investigation did not show any correspondence between the frequency of mistaken reactions and of sound associations; this want of correspondence has already been referred to by Weygandt and Miesemer. The action of alcohol varies from person to person, and from time to time, and involves now one form of mental activity, now another; in some individuals small doses produce motor excitement, while in others paralytic symptoms predominate. It is possible that individuals of the latter type, without increased motor activity, show a tendency to sound and phrase associations; such a combination is sometimes seen in quiet epileptics and catatonics, who may in addition to verbigeration show a marked tendency to sound-associations.

The length of reaction-time in reactions involving choice, the extent of variations in reaction time, mistaken reactions, sound associations and efficiency of perception stand in much more complex relations to each other than the results of previous investigators would indicate. C. MACFIE CAMPBELL.

ON DEGENERATIVE ECCENTRICITY. (Ueber degenerativ Vers-
(380) **chrobene.)** K. BIRNBAUM (of Herzberge), *Monatsschr. f. Psych. u. Neur.*, April 1907.

THE individuals who form the group discussed by the author are constitutionally peculiar and of poor balance, with a distorted judgment of values and an exaggerated idea of their own importance. Their interest is easily attracted to the novel and strange, both in science and philosophy; they tend to express themselves in a peculiarly elaborate and bizarre style, and frequently by eccentricities in their external appearance they seek to impose on others an adequate appreciation of their importance.

The mood is variable; the activity is usually superficial, and is confined more to words than to actual endeavours to modify the hard facts of existence. As their neighbours refuse to accept them at their own valuation and find their plans and ideas absurd, there is chronic friction and delusions that they are slighted in many ways. Unfortunate incidents may cause the quick development of systematised delusions, but these tend to simmer down. Such cases differ from cases of true paranoia in that there is no tendency to the slow formation of a fixed system of delusions. It is a permanently twisted attitude towards life, but not of a progressive

nature. These patients have always been grouped under paranoia. The differential diagnosis from constitutional mania is made on the absence of the unrest, of the hypomanic mood, and the flighty tendencies of the maniac.

The author gives the autobiographies of two cases.

C. MACFIE CAMPBELL.

TREATMENT.

CEREBRAL TUMOURS FROM THE POINT OF VIEW OF
(381) **OPERATION. (Les Tumeurs Cérébrales au point de vue du traitement operatoire.)** A. VAN GEHUCHTEN, *Le Névraxe*, Vol. ix., 1907, p. 69.

THIS is a record of a series of cases of cerebral tumours, several of which were subjected to surgical treatment.

1. *Tumour of left temporal lobe.*—Patient aged 39. At age of 30 he had been arrested for indecent exposure in public, but was found to be irresponsible for his actions. Nine years later, vomiting: attack of unconsciousness, with clonic spasms of the *left* face and limbs. Later, mental enfeeblement and difficulty in speech; attack of sudden bad taste in the mouth, with subjective disagreeable olfactory sensation, accompanied by an epigastric sensation, and followed by giddiness and occasional vomiting. Later, headache became localised to left temporal region and left-sided optic neuritis appeared, followed by paralysis of the left external rectus. A few days before death, there was an attack of convulsions in the left side of the face, whilst the right arm and leg became paresed, in addition to the limb of the left side. The deep reflexes were abolished on both sides; death from broncho-pneumonia. Autopsy showed a tumour of the left temporal lobe, chiefly in hippocampal convolution, compressing and displacing the mesencephalon to the right. The growth also implicated the lenticular nucleus and most of the convolution of the insula.

2. *Tumour of right occipital lobe.*—Patient aged 60. Three months before death he began to have attacks of unconsciousness, preceded by vertigo. No clonic movements were observed. About a month before death he was found on examination to have flaccid left-sided hemiplegia of arm and leg, without affection of face or tongue. The deep reflexes were exaggerated on the hemiplegic side; the plantar reflex was extensor. No affection of sensibility. The hemiplegia cleared up partially, but the patient gradually became weaker and died. At the autopsy a tumour was found in the

right occipital lobe in the region of the calcarine fissure. On inquiry it was then elicited from the patient's son-in-law that for three or four months before death the patient had been unable to see objects on the left side. There had evidently been complete hemanopia, which was not observed at the clinical examination (!). The cortico-spinal pyramidal fibres, by Marchi's method, were undegenerated; the hemiplegia was therefore due to compression of the brain stem, not to actual degeneration.

3. *Arachnoidal Cyst of right central convolutions.*—A patient, aged 28, developed attacks of subjective sensations of turgidity and intense pain in the right hand, ascending to the shoulder, and accompanied by loss of stereognostic perception in the hand. Sometimes the sensation spread to the right side of the face, and he fell unconscious, with clonic spasms of the right side of face and limb, micturition, and biting of the tongue. The right upper limb became feeble. The deep reflexes on the affected side were increased, but the plantar reflex remained flexor. Headache developed in the left temporal region, and the attacks of pain continued in the right arm. Operation revealed a large arachnoid cyst over the left central convolution, about its middle, containing clear fluid. The cyst was incised and drained, and the attacks of pain and convulsions ceased for a time. Later they reappeared, and the cyst was reopened and drained on numerous occasions, but only with temporary relief. Finally it was decided to resect the subjacent cortex to arrest the convulsions. This was done four and a half years after the original operation. The patient succumbed a few days later to meningo-encephalitis. At the autopsy a cyst was found in the region of the central convolution, both in front and behind the central fissure. The cyst communicated with the lateral ventricle. Old degeneration was found in the pyramidal tract by Pal's method.

4. *Multiple sarcomata of cranial bones.*—A boy of 12, who had a large sarcoma of the thoracic cage, developed multiple secondary growths on the cranial bones, causing intense headache. The patient was too dull mentally to permit of complete examination. At the autopsy, in addition to the tumours in the cranial bones, there were bilateral tumours in the middle fossa of the skull, compressing the tips of the temporal lobes. At the point of compression in each case there was an area of softening of the cortex.

5. *Glio-sarcoma of right motor area.*—A girl of 20 developed intense headache and vomiting and impairment of vision, about three months previous to coming under observation. Double optic neuritis had also been noted. Vision rapidly became impaired, until the patient was nearly blind, the right half of the visual field being much more affected than the left. Attacks

of trembling developed, without loss of consciousness, commencing in the left elbow, ascending to the left face, and spreading to the left lower limb. Operation revealed a cystic glio-sarcoma on the surface of the brain in the region of the right pre-central convolution. The growth was removed. The patient recovered from the operation without motor weakness of the limbs or abnormality of reflexes. The blindness, however, remained complete in the right eye. In the left eye, vision was reduced to a small area on the left half of the field; but after several months the right half of the field recovered to a slight extent. The visual defects were due to optic atrophy. PURVES STEWART.

THE DIAGNOSIS AND OPERABILITY OF BRAIN AND SPINAL
(382) **CORD TUMOURS, AND THE RESULTS OF OPERATION.**
(Zur Diagnostik der Operabilität der Hirn- und Rückenmarkstumoren und ueber Operationserfolge bei denselben.)
SCHULTZE, *Mittheil. aus d. Grenzgebiet. d. Med. u. Chir.*, Bd. XV., p. 613.

IN some cases the tumour may remain latent, or be masked by other clinical pictures, such as that of progressive arterio-sclerosis, with occasional cerebral hæmorrhage. All the symptoms of tumour may exist without its being present, as, for instance, in hæmatoma of the dura mater resulting from pachymeningitis. Choked discs were formerly regarded as pathognomonic of cerebral tumour, but Bruns and Stoelting believe that multiple sclerosis may produce choked discs, with cerebral symptoms. The differentiation from hydrocephalus internus is almost impossible. The diagnosis of hydrocephalus cannot be definitely established until the condition has persisted for a long period, though gummata or echinococcus may also remain stationary for years. Cases in which all the recognised symptoms, exclusive of choked discs, are present, but in which on post-mortem examination no obvious cause is revealed to account for the cerebral symptoms and also no hydrocephalus, are described by Nonne as pseudo-tumours. No cases of this nature have, however, come under the writer's observation. They are analogous to cases of pseudo-sclerosis, which imitate the symptoms of multiple or disseminate sclerosis. The correct diagnosis of cerebral tumours is of special importance from the point of view of surgical treatment. In cerebellar tumour headache is frequently localised in the frontal region, while it is often occipital if the tumour is situated in the frontal region. Choked discs may occur with frontal tumours, and it may be absent in cerebellar tumour, and bilateral anosmia may also be present in cerebellar tumour, owing to the secondary hydrocephalus causing pressure on

the nervi olfactorii. In such cases Neisser's puncture is often useful. Up to the present the Röntgen rays have only given a positive result in cases of tumour of the hypophysis, with resulting changes in the sella turcica. When it has been ascertained whether the tumour originates from the cerebral meninges or not, its operability or otherwise can be determined. Neisser's method may be of service in this connection, and in some cases the nature of the growth can be ascertained by this means. Psammoma usually originate from the membranes, though they may also develop intra-cranially. It is also desirable to obtain information as to the size of the tumour, and whether it is single or multiple. Operable cases should be dealt with as early as possible, as the smaller the growth the more favourable the prognosis of the operation. As regards the nature of the tumour, it is easy to ascertain by Neisser's method whether a cyst or abscess is present, but much more difficult in the case of glioma or sarcoma, as one can only aspirate small pieces of the growth. Gliomata are in most instances adherent to the substance of the brain, so that complete removal is impossible, the prognosis in these cases being especially grave in children. During the last fourteen years the writer has recommended operation in 19 out of 97 cases of cerebral tumour, usually only as a palliative measure. In one case only cure resulted. This was a glioma of the cerebellum, which was, however, easily enucleated. In another case a diagnosis of tumour of the left anterior central convolution was made. A tumour weighing 150 g. was enucleated, but the patient unfortunately died a few hours after the operation owing to considerable hæmorrhage. She was thirty-six, and much better results have been obtained in younger patients, especially by Victor Horsley, whose method of operation was described at the Meeting of the British Medical Association at Toronto. In 1906 Philip Coombs Knapp of Boston summarised 828 cases operated upon. Of these, 32 per cent. died soon after operation, and 54 per cent. were not improved, so that only 14 per cent. recovered, and cases of complete restoration to health are exceedingly few. He points out that removal of a portion of the brain results in impairment of the functions of the brain in a greater or lesser degree. In 3 only of the 19 cases operated upon by the writer has there been long-standing improvement. Almost all, with the exception of two or three, died immediately or shortly after operation. The estimation of the operability of these tumours varies between 3 and 17 per cent. Walton takes 4 per cent. as the average. In 8 cases examined post-mortem during the last year the writer has found two tumours which were technically operable. In one case the tumour was situated in the third right frontal convolution and in the other in the cerebellum. We can

now operate according to Scheide's and Horsley's methods on a comparatively large number of cerebellar tumours, excluding the relatively rare cases in which cysts are associated with gliomata. By Krause's method certain tumours at the base of the brain can be removed, especially in the pontine angles, and possibly also those of the hypophysis. Where it is impossible to localise the tumour, palliative operation should be performed at the earliest possible moment. Horsley recommends that medical treatment should not be tried for more than two to three months, and that intracranial pressure should be relieved by trephining if optic neuritis is present, and with this view the writer agrees. Improvement of the technique of the operation must also be considered, though even with the best possible surgical methods we can only hope to save or relieve a small proportion of the patients. The diagnosis must be made, the origin of the tumour ascertained, gliomata and sarcomata recognised, possibly with the aid of serum reactions, and means found to inhibit the growth of tumours similar to those adopted by Ehrlich in the case of sarcoma and carcinoma. It may in future be rendered possible to entirely remove tuberculous tumours, cysticercus, echinococcus, and gummata, by further investigations with regard to the ætiology of these growths. The prognosis of spinal cord tumours is dependent chiefly upon early and correct diagnosis, which is easy in typical cases, but in others difficult or impossible. As regards differential diagnosis, acute myelitis without trauma, syphilis, or acute infectious disease are extremely rare. Multiple sclerosis sometimes exhibits similar symptoms to those produced by tumour. The general symptoms of an operable tumour of the spinal cord are gradual development of motor and sensory paralysis of the extremities, extending up the trunk to a definite level, and slowly increasing for months and years, with inconsiderable variations in level and intensity. This is usually preceded by unilateral or bilateral neuralgia, the pain of which is increased by coughing or sneezing, or by rapid increase of pressure of spinal fluid. There is often localised pain over individual vertebræ, and not infrequently more or less severe Brown-Séquard paralysis, though in many instances radiating as well as localised pain is absent. The exclusion of tuberculous caries of the vertebral column has been rendered more easy by the X-rays, and this condition is also frequently accompanied by deformity and more rigidity of the spinal column. It is important to ascertain whether secondary metastatic tumours are present, as in these cases cure cannot be expected to result from operation, though the symptoms may be relieved. The diagnosis between intra-medullary and extra-medullary tumours is often difficult or impossible. The former, however, usually originate in the spinal membranes, the latter in the dorsal portion

of the cord, and partial sensory paralysis is frequently associated with intra-medullary tumour. In rare cases a circumscribed thickening of the dura mater resembling tumour, and leading to pachymeningitis chronica, may develop in the dorsal portion of the cord, and in these cases a history of syphilis may be suspected. Oppenheim has observed a circumscribed cystic meningitis, which clinically may be regarded as a kind of tumour. The statistics which we now possess with reference to the relationship of circumscribed sensory and motor disturbances to the individual segments of the cord are of great assistance in the localisation of tumours, as we are thereby enabled to operate without being compelled to remove too many vertebral arches. The diagnosis is usually most easily made in the dorsal and cervical portions, while it is most difficult in the cauda equina and conus terminalis. The prognosis of operation is most favourable in the dorsal region, and most unfavourable in the lumbar. The writer diagnosed the condition correctly in 14 cases, and in a fifteenth case, in which tumour of the spinal cord was diagnosed, autopsy revealed also the presence of multiple sclerosis. In one case a secondary sarcoma of the thyroid was found which could not be completely removed, while in another case a tumour-like pachymeningitic thickening was present. As regards the results of operation, out of 62 cases reported up to the beginning of 1905, there was cure or considerable improvement in 38 per cent.; while of 11 cases operated upon by the writer, 4 were completely cured and 1 greatly improved, which is a satisfactory result as compared with those obtained by operation on intra-cranial tumours. In a twelfth case there was an extra-dural tumour in the region of the 6th and 7th dorsal segments, which was successfully removed. In this case the symptoms of paralysis were unaccompanied by pain or localised sensitiveness to pressure. DONALD ARMOUR.

DIAGNOSIS AND OPERATIVE TREATMENT OF SPINAL CORD TUMOURS. (Zur Diagnostik und operativen Behandlung der Rückenmarkstumoren.) (383) SCHULTZE, *Münch. Med. Wochens.*, Juli 9, 1907, p. 1136.

THESE tumours usually have a neuralgic initial stage, but this is not invariably present. As an illustration, a case is given of a woman aged 21, who first experienced a feeling of weakness and numbness in both legs two years before she came under observation. There was never any pain. There was paræsthesia in the the legs, especially in the region of the knees, and numbness around the body, above the umbilicus, at about the level of the lower costal arch. The patient was admitted to the hospital on

November 18, 1906, and stated that in the middle of October the gait became unsteady and shuffling, and the legs stiff, the left leg being especially affected. There was also frequency of micturition. The family history was good. On examination there was found to be no nystagmus, intention tremor, or ataxia of hands. Abdominal reflexes absent; patellar reflexes active, but not abnormally so; moderate ankle clonus on both sides; plantar reflex absent; no Babinski's sign; coarse movements of leg muscles not materially diminished. Hypæsthesia and hypanalgesia up to about level of eighth dorsal segment; arms normal. Spinal column mobile; no pain on pressure. A diagnosis was made of multiple sclerosis or meningeal tumour, and bathing with hot water was recommended, together with the administration of iodide of sodium. On January 3rd, 1907, the patient walked into the consulting-room, but when lying in the abdominal position suddenly became unable to walk or stand, sensibility being unaffected, and pain still absent. The course of the illness strongly suggested the presence of a tumour of the medulla spinalis. The patient stated that micturition was often delayed. Pain in the back had only appeared recently, and only after special exertion; there were no radiating pains. She was able to walk a few steps with difficulty when supported on both sides. Severe spastic paraparesis with ataxia; no distinct nystagmus; arms and hands normal; could not rise from the recumbent position without assistance; cranial nerves normal; no change in fundus oculi. Tendon reflexes remained the same in the arms; normal ankle clonus on both sides, but there was no Babinski's sign on the left, and no plantar reflex on the right side. Hypæsthesia on both sides for all kinds of sensation up to the level of the xyphoid process, somewhat more pronounced below the knee on the right side than on the left, and extending for about 2 cm. above this on the left side was a hyperæsthetic zone. Sense of position considerably impaired on both sides; spontaneous muscular spasms affecting both legs, especially the left. The spinal column was normal, and there was no circumscribed sensibility to pressure. The motor paralysis increased, and walking became impossible. The patient wished for operation, which was performed on February 16th. The diagnosis of multiple sclerosis was excluded by the course and symptoms. The gradual progress and want of a known cause was against chronic myelitis, the absence of definite pain, and increased intensity of symptoms against that of chronic meningo-myelitis. The rapid increase of motor symptoms when the patient was in the abdominal position may be explained by an increase of pressure, especially as there was no sensory disturbance. Recent researches confirm the influence of the horizontal or nearly horizontal position upon the paralysis in these cases.

In none of the organs of the body could the presence of tuberculosis or of new growth be demonstrated. Syphilis was excluded by the result of examination, the course of the illness, and the complete failure of iodide of sodium, and there was no history of rigidity or loss of sensation in the column. Intramedullary tumour is comparatively rare in the dorsal portion of the cord, and dissociation paralysis was absent. The writer has twice observed such complete absence of pain and sensibility to pressure, and a similar case is reported by Oppenheim. In one of Schultze's cases the tumour was of the size of a hazel-nut. In another case a large tumour, 1·5 cm. in length, was found on the right side, so that persistent absence of pain is not a contra-indication for the performance of laminectomy. In the case under consideration, an extra-dural tumour about the size of a walnut, situated posteriorly and to the left, opposite the fourth and fifth spinous processes, was removed, and proved to be a fibroma. No elevation of temperature ensued, and the motor and sensory symptoms rapidly improved. The writer has observed no trace of hysteria or of hysterical analgesia in any case of this kind which has come under his observation. He also reports the case of a man aged 56, who complained of irritation in the legs and painful sensations in the right shoulder. A diagnosis of compression, with a probability of extramedullary tumour, was made, and laminectomy performed. When admitted to the hospital in November 1906, the patient stated that at the beginning of September there was a "tight band" sensation from the inguinal region to the pit of the stomach. Subsequently motor weakness of the legs developed, but no sensory symptoms. On admission there was spastic paraplegia of the legs, whilst sensibility was normal, the fifth thoracic vertebra being sensitive to pressure. There was nothing abnormal in the cord or lungs, and the urine was normal. There was a painful girdle sensation at about the level of the seventh dorsal segment. Compression of the medulla spinalis was the most probable diagnosis, based, as in the former case, upon the complete loss of sensation in association with severe motor paralysis, the pain on pressure over the fifth thoracic spinous process, with hyperæsthesia at the level of the girdle sensation in the neighbourhood of the seventh dorsal segment, indicating that the site of the compression was opposite that process. Intramedullary tumour was rendered improbable by the sharp localised and radiating pains, while the intermittent hypæsthesia was against the diagnosis of extramedullary tumour. At the operation no tumour was found, and there was no escape of cerebro-spinal fluid. The spinal membrane was thinner than normal. At the end of December (a fortnight after the operation) cystitis appeared, and in February an abscess developed in the scar of the operation wound, the patient dying

on March 21st. On section, there was necrosis of the fourth and fifth thoracic vertebræ and the adjacent heads of the ribs, a large ichorous abscess in the posterior mediastinum, and a circumscribed gangrenous focus in the apex of the left lung. There was no tuberculosis or carcinoma of the œsophagus. The disease appeared to have developed in the cord, associated later with changes in the lungs. The dura mater was much thickened at the level of the affected vertebræ. Macroscopically the spinal cord showed degeneration of the lateral and posterior columns, and descending degeneration of the pyramidal tracts. From eighteen cases collected by the writer, he concludes: (1) A correct diagnosis is often not difficult, as many cases of dorsal spinal-cord tumours run a typical course; and (2) in most cases operation on the dorsal portion of the cord is without risk, especially when it is unnecessary to open the dura. In most of these cases extra-medullary tumour was probable, and as regards paralysis the results of operation were satisfactory. Three of the cases were not operated upon, and in two no tumour was found, either on operation or on section. In one case the tumour could not be found at the operation on account of its being situated high up. Of 13 cases of meningeal tumour operated upon, 6 completely recovered and 1 was permanently relieved. In 9 cases of tumour operated upon by Oppenheim, cure resulted in 4, and in 5 further cases, diagnosed by him as extra-medullary tumour, no new growth was found. The writer's experience shows that the malignant tumours most favourable for operation are those situated opposite the dorsal cord. Of 8 patients with benign circumscribed tumours in this position, 6 were cured and 2 permanently relieved. In one case the tumour could not be completely removed. Cases of tumour-like pachymeningitis and circumscribed cystic meningitis, such as those reported by F. Krause, have never come under his observation. DONALD ARMOUR.

Reviews

THE NERVOUS SYSTEM OF VERTEBRATES. J. B. JOHNSTON. John Murray, London, 1907. Price 15s.

THE complex structure and the manifold functions of the nervous system make it imperative to employ all possible means of study which can throw light on the nature and methods of its activities. Till within the last few years the investigation, of the central

nervous system at least, has been restricted almost entirely to the higher vertebrates in which the structure is most complex and in which the functions are most highly differentiated. This has been but natural, as our more immediate aim has been to learn of ourselves, of the nature of our actions and activities, of the structures directly concerned in their production and of the nature and position of lesions which may lead to abolition or perversion of their functions.

But rich in results as this line of investigation has been it must be evident that that method which begins with the study of the nervous system in its simplest and least complicated forms and traces the correlation of structure increasing in complexity with the appearance of new or more highly differentiated functions can promise much in the study of the phenomena of life and of that on which the behaviour of an animal to its surroundings depends. This is the method adopted in Professor Johnston's volume; its aim is to give an account of the nervous system as a whole, to trace its phylogenetic history and to show the factors which have determined the course of its evolution.

Starting with the lowest vertebrates it is seen that the primary components of the nervous system are the somatic sensory and somatic motor systems, through which the animal is influenced by and responds to changes in its surroundings, and the visceral sensory and visceral motor systems which are concerned in its internal activities; but in even the simplest forms the existence of secondary sensory centres and the anatomical connections which make the combination of the different systems in function possible has produced a complex mechanism. It is by the modification and elaboration of these systems and of their central connections that the central nervous system of the higher vertebrates is derived; the aim of the comparative method of study is to ascertain the nature of its integral parts by tracing their gradual evolution. That there must be considerable danger of error in attempting to determine the functions of tracts or of centres by their connections or by their structure alone, even when guided by the history of their development, must be evident. Into such error it seems the author has fallen in including Clarke's column, the dorsal spino-cerebellar tract and its end nucleus in the vermis of the cerebellum in the visceral afferent division; as such these would represent the central sensory sympathetic system. The pages devoted to the cerebellum contain much valuable information; the view is expressed that the cerebellum represents a highly specialised part of the somatic sensory system, but in the fishes, at least, it contains a visceral sensory (gustatory) nucleus. Though from analogy with the better known mammalian cerebellum the inclusion of this visceral component may seem improbable, it must

be remembered that in the aquatic vertebrates a great part of the afferent impulses which bring about elaborately co-ordinated actions must be gustatory in nature.

The central connections of the visceral system can be traced more easily in fishes than in the higher vertebrates. In this connection it is interesting that it has been shown that in this class the taste buds, wherever situated, are always innervated by the visceral afferent roots of the cranial nerves which terminate in the visceral lobe; the latter is, in part at least, represented by the fasciculus solitarius of mammals. The central portion of the visceral efferent division is identified with that column of grey matter in the cord which is undoubtedly homologous with the intermediolateral tract.

The chapter on the evolution of the cerebral hemispheres is one of the most interesting and instructive in the book, though that devoted to the neopallium, as might be expected from the direction from which the subject has been approached, is perhaps the least satisfactory.

The nomenclature adopted by the author is simple and intelligible, and his pages are written in a concise and easy style.

The book can be as warmly recommended to those to whom the anatomy of the nervous system is of interest as the basis of its function, as to those whose interest lies in the study of morphology alone.

GORDON HOLMES.

INVESTIGATIONS INTO THE ETIOLOGY OF MANIA, PERIODIC MANIA, AND CIRCULAR INSANITY. (Untersuchungen über die Aetiologie der Manie, der periodischen Manie, und des zirkulären Irreseins.) By Dr G. SAIZ. Pp. 220. Berlin: S. Karger, 1907. Price M. 5.60.

THIS is an exhaustive statistical inquiry into the causes of mania of various kinds. The material for it has been furnished by the cases admitted into the Imperial Charité at Berlin for a little over eleven years (1895-1906). Of these there were 24,705, the male patients numbering 16,123, and the female 8582. From these the cases of mania have been extracted, and the causes of the mental disease investigated as fully as possible. The varieties of mania adopted as a result of this inquiry are four in number—simple, recurrent, periodic, and circular. The chief points the author had in view were to ascertain the frequency of the periodic and recurrent varieties, and whether periodic mania has a special etiology. An important preliminary statement is made as to the procedure adopted in judging to which class each case was to be referred. As between recurrent and periodic mania, by the latter is meant a

case which exhibits a more or less regular appearance of maniacal attacks, which are due to very slight external causes, or even to none at all. They are a manifestation of endogenous causes chiefly. Recurrent mania, on the other hand, is the result usually of distinct external causes of weighty import, the attacks are at irregular and, as a rule, prolonged intervals, and the intermediate period is one of complete sanity. It is acknowledged, however, that there are cases showing every grade of transition between these two forms, and that no hard and fast line can be drawn between them. A period of ten years is taken to distinguish between the two forms, but a better guide is the study of causation. If the same set of causes produce an attack, the case may be regarded as a recurrent one. If the above limit of time be taken, it consequently follows that the same person cannot have many recurrences during his lifetime. Owing to the arrangements under which patients are sent to the Berlin Charité the author, in the case of very many of them, saw them only once and not for a long period, so that it is not to be wondered at that the majority of the cases of mania are found in the class having only one attack. The conclusions that might be drawn regarding this group he therefore admits would not be quite reliable, and hence he puts them along with cases of recurrent mania in his statistical inquiry.

He further enters into the consideration of what is meant by the term mania, and adopts Ziehen's views. The two cardinal symptoms are emotional elevation and increased rapidity in the association of ideas. Cases in which hallucinations and delusions are prominent and are the cause of maniacal symptoms are regarded as acute hallucinatory paranoia. In this respect his statistics do not compare with others, such as those of Wernicke and Kraepelin, where this distinction has not been made. A historical retrospect occupies a good many pages before he reaches the subject proper of his thesis.

Coming to this we find that, of the 24,705 cases investigated, 88 (0·35 per cent.) suffered from simple mania. Of these 19 were men and 69 women. Only 1 man and 4 women were cases of recurrent mania. The reasons for the small number of these are given above. Of periodic mania there were 51 cases (0·21 per cent.) 8 being men and 43 women. Circular insanity showed 74 cases (0·29 per cent.) 24 men and 50 women. It will be seen from these figures that, in all forms of mania, the women supply the greater number of cases. He concludes from further analysis that this predisposition is due to endogenous causes not dependent on the difference of sex. Further particulars are given with regard to various other facts with respect to causation, such as age, civil state, occupation, nationality, heredity, head injury, mental stress, menstruation, pregnancy, the puerperium, acute infectious diseases,

and other physical disorders. These, however, cannot be summarised. His general conclusion is that in most cases several etiological factors, predisposing and determining, co-operate. There are, however, quite a number of cases in which no cause at all could be ascertained. Many interesting details are given by the author as a result of his inquiry, but these cannot be specifically referred to. No attempt is made to express the results of his investigation in mathematical terms, but the figures would furnish a useful basis for such work, and the results would be exceedingly useful. JAS. MIDDLEMASS.

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Review
of
Neurology and Psychiatry

Original Articles

THE EXACT DETERMINATION OF AREAS OF ALTERED SENSIBILITY.

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IN the determination of areas of cutaneous anæsthesia, experience may be taken now definitely to have shown that there are three methods of practical value; they are the use of cotton-wool as recommended by Head, the use of a very fine camel's-hair brush, and lastly, v. Frey's hair æsthesiometer. In the first two of these methods the touch is made with a very yielding material, it being assumed that the pressure exercised never exceeds that minimum which is adequate to produce a sense of contact on normal skin. It is clear, however, that in practice it is difficult to conform with this requirement. This fact may perhaps be one element in the difficulty which is often found in obtaining constant and precise boundaries to an anæsthetic area. With v. Frey's hair æsthesiometer the touches are made with hairs graduated to exercise different known pressures, and thus is avoided the variability of other methods. The method is based

on the not wholly incontestable conceptions that tactile sensation is aroused by pressure and pressure alone, and that light pressure without movement stimulates the same end organs as does light pressure with movement. None of the methods makes allowance for the variations of tactile acuity of different parts of the normal skin. This difficulty is for the most part in practice only encountered in v. Frey's method, and can there be met to a great extent by comparison of symmetrical parts; that it is not so conspicuous when the testing is done with cotton-wool or the camel's-hair brush merely shows that these methods are less delicate.

To these theoretical objections to the methods mentioned above may be added certain practical disadvantages.

(1) The difficulty of obtaining exact outlines. To everyone who has attempted to define precisely an area of tactile anæsthesia it must be known how greatly the responses of the patient to touches vary according to whether the boundary is being approached from the anæsthetic or from the normal skin; discrepancies of half an inch to an inch are common, and of as much as two inches not at all unusual.

A further difficulty in obtaining exact results is due to the frequency with which are felt purely hallucinatory touches. These are undoubtedly more common in recent cases of nerve injury than in old ones, and in conditions of partial destruction of conductivity such as tabes. Such hallucinations may be so frequent that almost necessarily they sometimes deceive the investigator. While it is probable that the tendency is for them to be felt more frequently in areas of complete anæsthesia, it is clear that they will be most deceptive when they are felt in areas of partial anæsthesia or while such areas are being investigated. Moreover, it has seemed to us that even the very slight pressure element in a succession of cotton-wool or camel's-hair touches over an anæsthetic area produces, by a kind of summation of stimuli, a sensation that in the end is described as a touch and may prove very confusing to the investigator.

These difficulties are due to the necessity of concealing from the patient the situation and time of the stimulus, and it is clear that the state of expectant attention which such conditions involve brings with it disadvantages which subtract considerably from the objectivity of the results.

(2) Difficulty in defining areas showing different degrees of

loss. Of the three ordinary methods it is clear that v. Frey's is the only one by which any attempt towards quantitative estimation of varying degrees of tactile loss can be made. When we bear in mind how common is relative anæsthesia, and how it includes all stages of loss from the just not normal to the almost complete tactile anæsthesia, we cannot but realise that there must be distinguishable steps in this wide range, the discovery of which would be important in clinical medicine if it could be made readily.

(3) The difficulty in finding and outlining areas of very slight loss. It seems often to be assumed that areas of relative anæsthesia are essentially areas of vague and indefinable outline. Now it is clear from very little consideration that any change of sensibility resulting from a nerve lesion is a perfectly definite thing with a perfectly definite anatomical distribution, and that the apparent vagueness and comparative clinical uselessness of areas of slight change are merely evidences of the grossness of our methods of examination. There can be no doubt that if we could examine such areas satisfactorily we should find the distribution of them just as valuable in diagnosis as are areas of complete tactile anæsthesia, with the additional advantage of obtaining precise information quite early in the history of the case.

In the course of the examination of a number of cases which showed cutaneous tactile anæsthesia, we soon became convinced that there was a considerable discrepancy between the areas beyond which the response to cotton-wool was absolutely normal in every way, and the areas which were felt by the patient not to be normal. We at first assumed that these marginal areas were indefinable, but we ultimately found that they could be outlined with considerable exactitude. The existence, then, of sensory changes totally insusceptible of detection by cotton-wool touches is one of the most serious drawbacks from the value of that method in early diagnosis.

In attempting to obtain some improvement in the resources of the clinician in dealing with tactile anæsthesia three facts need consideration. First, the patient may be conscious when he touches his own skin of an abnormality of sensation too fine to be detected by any known objective method. Secondly, the attitude of expectant attention rendered necessary in the use of any such method involves fallacies which may be very misleading.

Thirdly, many of the difficulties in the application of these methods seem to be associated with the attempt to apply a uniform minimal pressure. Consideration of these facts led us to inquire whether the patient himself could not outline the maximal area of sensory change with a satisfactory degree of precision. We have found this to be in fact very often the case.

The patient is asked to explore the suspected area with his finger-tip and to mark where he feels lines of transition to occur, looking at the part and using any degree of lightness of touches which enables him to make the distinction. It will be found that an intelligent patient soon discovers that, by trying various lightnesses of touch and approaching the suspected boundary from different directions, he can finally settle on a continuous outer limit with great certainty, and will probably be able with equal certainty to detect within this margin subsidiary areas of different sensory quality. We refer for convenience to this little device for investigating cutaneous anæsthesia as the "stroking method." It will be seen that it makes use of the patient's own sensation of the abnormal, that it avoids the troubles due to expectant attention, and that it gets rid of the difficulties incident to the minimal pressure methods by including always a very large pressure element which is therefore relatively constant. It is obvious that its advantages are most marked in the investigation of areas not entirely insensitive to cotton-wool, and in the analysis of such areas into districts of different degrees of tactile change. The maximal area obtained by this method is bounded by a margin which is constant, sharply defined, and as a rule of strikingly simple form. It contains within it all the sensory changes produced by the nerve lesion—analgesia, thermanæsthesia, tactile anæsthesia, hypoæsthesia, and, in addition to these, a marginal area more or less wide in which no form of sensory disturbance other than an alteration in quality can be detected.

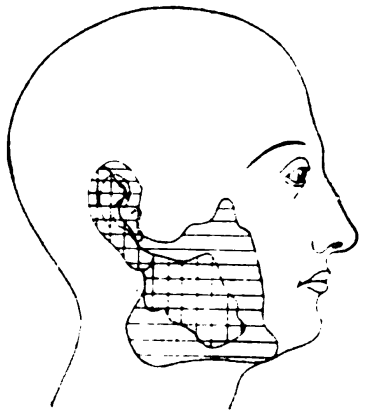

FIG. 1.

Vertical shading denotes anæsthesia to camel's-hair brush.
Horizontal shading denotes area abnormal to stroking touch.

CASE 1.—Tactile changes following section of great auricular nerve. Examination twenty-four hours after section.

The significance of this last-named sensory change we shall in the near future deal with elsewhere.

The following cases are selected from a considerable number which were all met with within the last few weeks and will serve to illustrate in what directions the method may be found of practical usefulness.

CASE 1. *Section of great auricular nerve. Area defined by stroking method much larger than area defined by cotton-wool.*

This patient was a man in whom the great auricular had been divided at the posterior border of the sterno-mastoid during the course of an operation on the neck. The diagram (Fig. 1), which gives the results of an examination made within twenty-four hours of the section of the nerve, shows how much larger the area defined by the stroking method was than that within which touches with the camel's-hair brush could not be felt; except on the ear the separation between the two lines is in general as much as an inch wide. This is of some interest anatomically, since one of us[1] has shown that the area of distribution of the fifth nerve, as determined by the camel's-hair brush after section, never comes below the level of the tragus. If, then, this method of estimating anæsthesia were relied on alone it would appear that the areas of distribution of the two nerves do not meet.

CASE 2. *Extensive injury to cervical plexus. Loss of sensation as defined by camel's-hair brush confined to area of great auricular nerve.*

The patient was a young woman admitted to the National Hospital, Queen Square, under the care of Dr James Taylor, to whom we are indebted for permission to make use of the notes. Fifteen months earlier she had undergone an operation for wide-spread tuberculosis of the glands of the neck. At the time of admission she showed anæsthesia as determined by the camel's-hair brush in a part of the area of the great auricular nerve only. Investigation by the stroking method, however, demonstrated a relative anæsthesia of the whole of the great auricular

[1] H. Morriston Davies, "The Functions of the Trigeminal Nerve," *Brain*, Part ii., 1907.

(*cf.* diagram of Case 1), the superficial cervical, the supra-sternal, and the small occipital branches of the cervical plexus (Fig. 2). The case is included because it shows how very great may be the disproportion between the areas of defective sensation which may be detected by the two methods. It was at first not suspected that more than a mere fringe of relative tactile anæsthesia surrounded the complete, and therefore there was

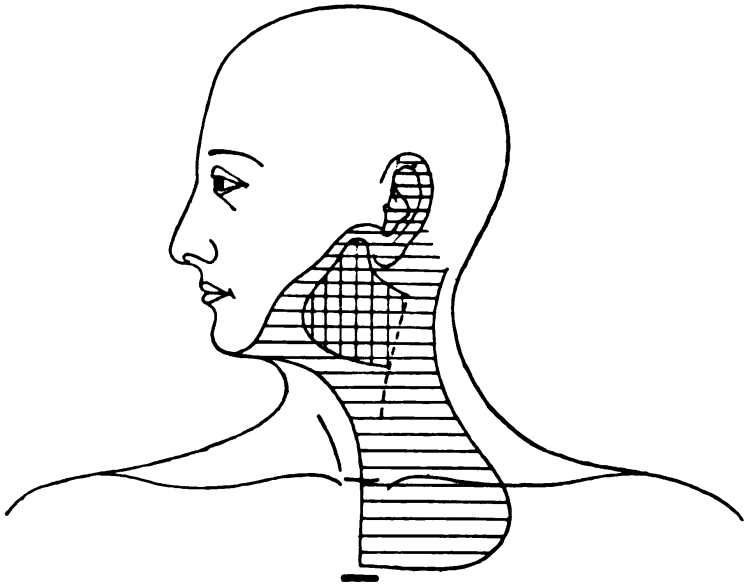

FIG. 2.

Vertical shading denotes anæsthesia to camel's-hair brush.
Horizontal shading denotes area abnormal to stroking touch.
Broken line denotes scar.

CASE 2.—Tactile changes fifteen months after extensive injury to cervical plexus. Camel's-hair anæsthesia in area of great auricular nerve only. Abnormality to stroking touch in areas of superficial, cervical, suprasternal, and small occipital nerves, and whole of great auricular. Sensory loss behind ear could not be investigated beyond the margin of the hair.

nothing to suggest how extensive had been the injury to the cervical plexus from which the patient was recovering.

CASE 3. *Cervical rib. No anæsthesia to cotton-wool. Altered sensibility in area of eighth cervical nerve.*

The patient, a married woman aged 27, was admitted to University College Hospital under the care of one of us. She complained of lumps in the right side of the neck, and that she had been subject to pains in the right arm; these latter passed

along the whole length of the limb into the middle finger; she had had them for some years and had been told that they were due to rheumatism. Examination showed a number of greatly enlarged hard glands deeply placed behind the lower end of the right sterno-mastoid, and the presence of a bony mass, obviously a cervical rib, in each posterior triangle. There was no evidence of pressure on the subclavian artery, no localised atrophy or

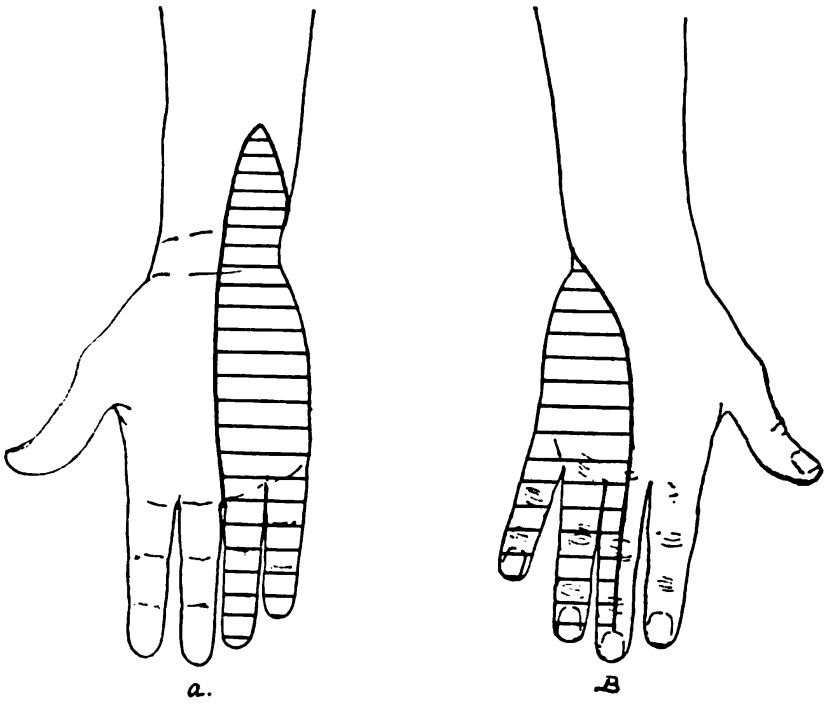

FIG. 3.

Shading denotes area abnormal to stroking touch.

CASE 3.—Cervical rib. The root distribution of the tactile disturbance discovered by stroking touch is well shown. The whole of this area responded normally to cotton-wool.

A. Anterior aspect. *B.* Posterior aspect.

weakness of muscle, and no loss of sensation to cotton-wool in the whole of the right upper limb. Investigation by the stroking method, however, revealed a very definitely outlined area of altered sensibility, having the distribution shown in the diagram, and corresponding with that of the eighth cervical nerve (Fig. 3).

The enlarged glands and the cervical rib on the right side were removed. It was found that the lower parts of the roots of the brachial plexus were pressed against the subclavian artery

by the rib, and on account of the shape of the rib it seemed that the eighth cervical nerve had been subjected to a greater degree of compression than had the first dorsal. Within a few days

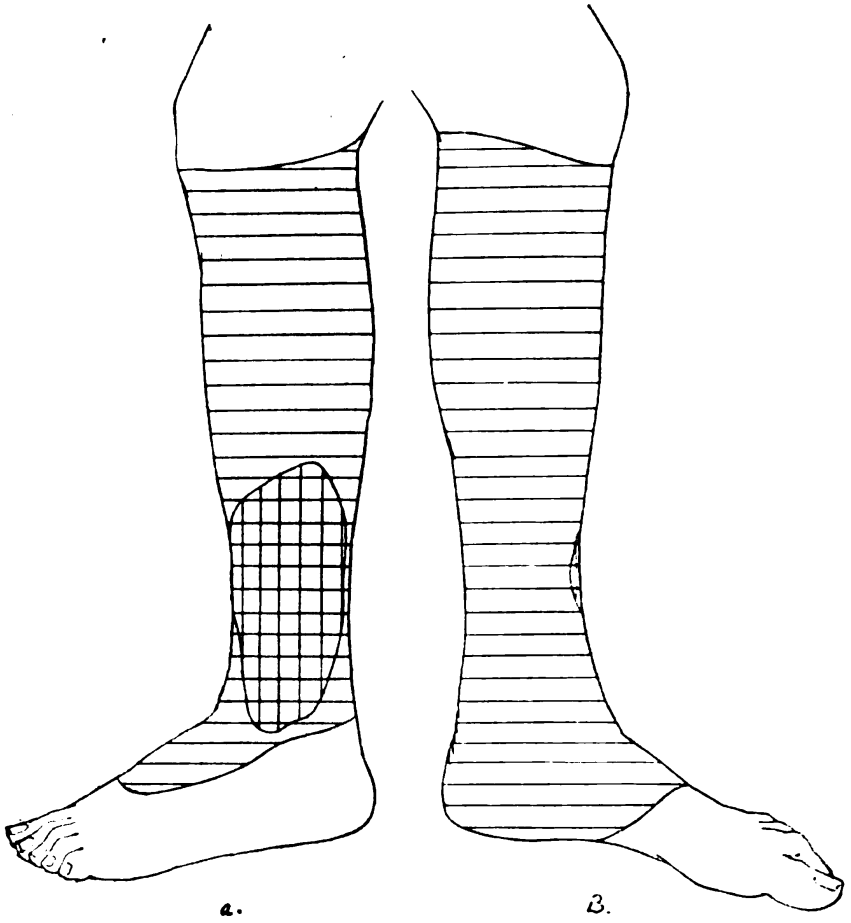

FIG. 4.

Vertical shading denotes anæsthesia to cotton-wool.
Horizontal shading denotes area abnormal to stroking touch.

CASE 4.—Lesion of sacral plexus. Cotton-wool anæsthesia contained within area of external popliteal. The area defined by stroking touch showed that, in fact, the lesion was a very extensive one.

A. External aspect. *B*. Internal aspect.

after the operation the area of altered sensibility had completely disappeared.

In this case the sole objective sign that the rib had begun to compress the plexus could only have deen detected by the method that was employed. From the form of the area of sensory change it was at once obvious that a root of the plexus and neither the plexus itself nor any one of its branches was

being involved. This made it highly probable that the glands were not the source of the pain. It was clearly of very great advantage to be able to make the diagnosis that there was organic involvement of the plexus at so very early a stage of the condition.

CASE 4. *Diagnosis of lesion of sacral plexus from lesion of external popliteal by outline of area of sensory change.*

The patient was a woman aged 30, under the care of Dr Ernest Jones, to whom we are indebted for the opportunity of examining her. She had been suffering for some months from symptoms suggesting chronic pelvic cellulitis on the left side. There was much pain extending throughout the whole length of the left lower limb, but no obvious weakness or wasting of muscles. On the middle of the back of the thigh, in its upper half, there was a deeply depressed longitudinal scar exactly over the course of the great sciatic nerve and firmly attached to the femur. It was the result of two operations in childhood for disease of the bone. Examination of sensibility showed on the outer side of the leg an area of insensibility to cotton-wool touches (see diagram, Fig. 4).

Although the general features of the case pointed to a lesion of the sacral plexus, the presence of the scar at the back of the thigh made quite plausible the supposition that the anæsthesia might be due to an involvement there of the external popliteal part of the great sciatic nerve. It was clear, however, that there was a considerable area of altered sensibility beyond the anæsthesia, and an exact determination of this by the stroking method showed that it was of such a distribution as to be incompatible with a lesion of the great sciatic (Fig. 4).

The case shows well the value, in diagnosis, of being able to define exactly areas of sensory change not susceptible of determination by the usual objective methods.

CASE 5. *Lesion of the fifth cranial and great auricular nerves showing dissection of sensory change into four anatomical areas.*

The patient was a man aged 32, admitted into the National Hospital, Queen Square, under Dr Ferrier, to whom we are

indebted for permission to refer to certain aspects of the case. The man was suffering from headache, vomiting, and optic neuritis; he had in addition attacks of pain in the areas of the fifth cranial and great auricular nerves on the right side. There was no weakness of the motor part of the fifth. Examination of the sensibility of the face by the camel's-hair brush showed slight relative anæsthesia over the ear and lower jaw. This anæsthesia had extremely indefinite boundaries. Over the temple and forehead all touches were recognised. The patient was asked to attempt to define with his own finger what areas of abnormal skin he felt. He at first marked out an area corresponding exactly with that of the cutaneous distribution of the great auricular and second and third divisions of the fifth. He was then asked if the sensation given by this area was the same all over it, and he was now able to distinguish three grades of altered sensibility, which he mapped out with great precision: these areas corresponded with the areas of the great auricular nerve, where the loss was most marked, the inferior maxillary division of the fifth and the superior, where the loss was least marked. Further exploration of the face by the stroking method led him to detect a slight difference between the two sides of the forehead: he thereupon marked out with striking nicety the area of supply of the first division of the fifth (Fig. 5).

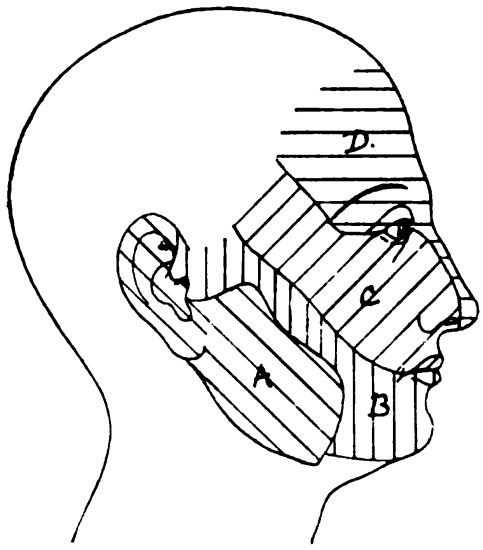

FIG. 5.

CASE 5.—Intracranial tumour. Diagram shows four areas of tactile change of different degrees of intensity discriminated by patient by stroking touch.

A. Great auricular nerve; change most marked.
B. Inferior maxillary nerve; change less marked than in *A*, more marked than in *C*.
C. Superior maxillary nerve; change less marked than in *B*.
D. Ophthalmic nerve; change very slight.
Partial loss to cotton-wool in *A* and *B*. Investigation could not be carried beyond margin of hair.

It is interesting to note that a distinction similar to that between the sensations felt in the cutaneous areas of the second and third divisions was perceptible on the mucous membranes.

The whole of the mouth on the right side felt abnormal to the patient, and the change was more marked on the tongue, the lower gums, and the cheek, than on the hard palate and upper gums.

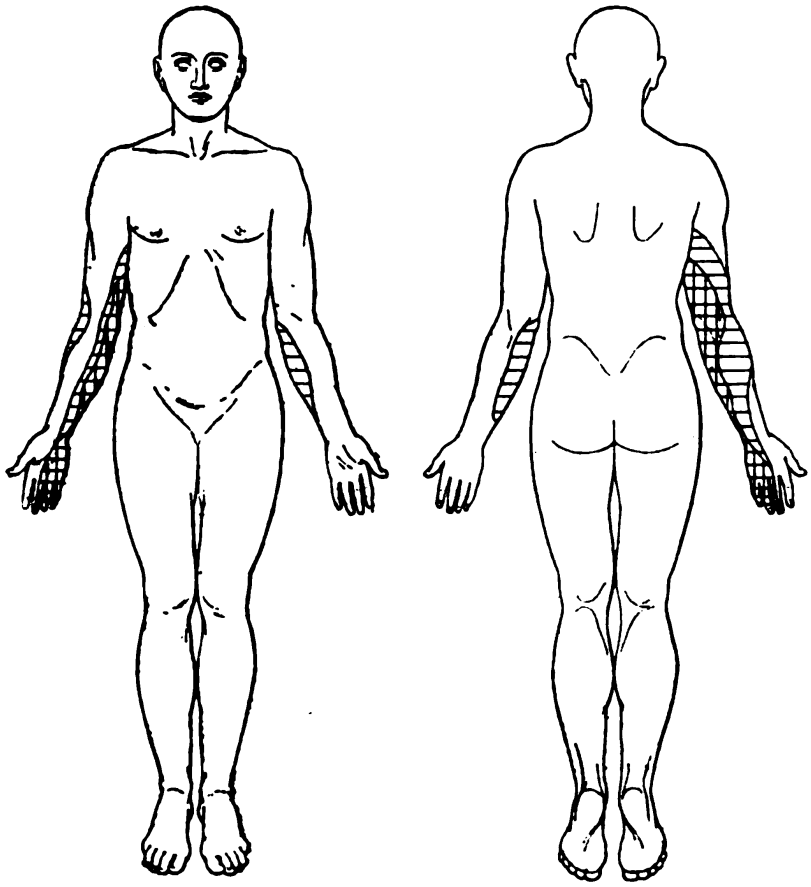

FIG. 6.

Vertical shading denotes anæsthesia to cotton-wool touches.
Horizontal shading denotes area abnormal to stroking touch.

CASE 6.—Pressure on cervical and dorsal nerve roots. On the right side there is tactile anæsthesia in the areas of the eighth cervical, and first and second dorsal nerves. On the left side perception of cotton-wool touches is perfect, but stroking touch shows that the first dorsal nerve is already beginning to be involved.

This remarkable capacity to distinguish amongst areas of altered sensibility of different degree with such exactitude as to give results so strikingly corresponding with the anatomical distribution of the various nerves must undoubtedly be attributed chiefly to the exceptional intelligence of the patient, and, to some extent, to the sensitiveness normally of the part concerned.

CASE 6. *Pressure on spinal nerve roots. Tactile anæsthesia in right arm, altered sensibility in left.*

We are indebted to Dr Ernest Jones for opportunities of investigating this case.

The patient, a woman aged 63, presented evidences of a growth, probably a secondary carcinoma, compressing the lower cervical and upper dorsal nerve roots. She had had pains in the right upper limb for four months, and during the last six weeks had noticed wasting and weakness of the limb, especially in the hand. She had felt numbness and tingling in the right forearm for some weeks, and in the left for a fortnight. There was marked wasting in the right arm of the muscles supplied by the sixth, seventh, and eighth cervical, and first dorsal nerves. Wasting of similar distribution but less degree was present on the left side.

Anæsthesia to touches with cotton-wool was found on the right side in the areas supplied by the eighth cervical and first and second dorsal nerves. This anæsthesia was surrounded by the usual area of altered sensibility to stroking touch.

On the left side examination revealed no hypoæsthesia whatever, but by the stroking method an area of altered sensibility corresponding with the distribution of the first dorsal nerve was readily made out (Fig. 6).

A fortnight later the patient was examined again, and it was found that this area was beginning now to be partially anæsthetic to cotton-wool touches; the area of change of sensibility to stroking touch had, however, extended to the distribution of the eighth cervical nerve, which it exactly outlined.

The case is referred to here as it shows very well the three striking features of isolated change in sensibility to stroking touch, viz. that it is next to pain the first sensory manifestation of compression of a nerve, that it corresponds with the anatomical distribution of the nerve, and that it precedes by a definite period any objective evidence of anæsthesia.

CASE OF TRIGEMINAL NÆVUS, ASSOCIATED WITH EPILEPSY AND CONTRA-LATERAL HEMIPARESIS.

By J. W. STRUTHERS, F.R.C.S.Ed.,
Assistant-Surgeon to Leith Hospital.

IN a paper, of which an abstract appeared in the last issue of this journal, Cushing relates three cases in which nævi, corresponding more or less closely to the distribution of the fifth nerve on the face, were associated with epilepsy, etc. In two of the cases the superficial nævus was found to be associated with a nævoid condition of the dura mater on the same side, which had given rise to subdural hæmorrhage, this in turn having caused the epilepsy. From the similarity in the history and clinical features, Cushing inferred that a like condition was present in his third case.

In one of his cases marked benefit followed operation, with separation of cortico-dural adhesions, after a preliminary ligation of the external carotid artery to prevent undue loss of blood from the vascular dura mater. Encouraged by this report the writer was led to operate on a case recently under his care, with a strikingly similar history and condition, in the hope of benefiting an otherwise apparently incurable epileptic. The case is recorded to show that other morbid conditions may give rise to a precisely similar clinical picture to that recorded by Cushing, and that the presence of a trigeminal nævus on the same side as a presumable intracranial lesion in such cases does not necessarily imply any direct relationship between the two.

D. W., æt. 14 years, was admitted to Leith Hospital on June 3rd, 1907. According to his mother's somewhat incomplete statement, he was born with a large nævus, extending over the left side of the head and face. There was nothing abnormal in the labour or birth, and no instruments were used. Till eight months old he was healthy. In the ninth month, without any assignable cause, he was seized with a convulsion which affected his right arm and leg, and left them weak. During the next ten months he had four similar seizures. Except for a permanent weakness of the right side, most marked in the right arm, he remained well till his eleventh year, when the fits returned again without obvious cause, and

he continued to suffer from fits at frequent intervals up to the time of admission. He sometimes had several fits in one day, sometimes was free for a fortnight. During the fits he lost consciousness and jerked all over, but mostly on the right side. He had been treated for his fits, presumably with bromides, without benefit.

There were no other material facts in the history.

On examination.—The lad was found fairly well developed, but rather undersized for his age.

On the left side of the face and head there was a well marked cutaneous nævus extending over the distribution of the upper, middle, and greater part of the lower divisions of the 5th nerve. It extended very slightly across the middle line on the forehead and nose. The mucous membrane of the left side of the nose and the left half of the soft palate were unduly vascular, and the left conjunctiva and iris were also nævoid. There was also a cataract of the left eye, apparently the result of intraocular hæmorrhage, and the boy had no perception of light in the left eye. Considering his upbringing, the boy's intelligence was fair, he could read and sing, but could not write. He answered intelligently, but his speech was deliberate and slow, though otherwise unaffected. Could his fits have been cured, he would have made a useful member of society, and been able to earn his living.

In walking he dragged his right leg a little, owing to a slightly spastic condition of the limb. The muscular power of the right leg was markedly weaker than that of the left, the calf and thigh measuring half and three-quarters of an inch less respectively on the right than on the left side.

He made very little voluntary use of the right arm and hand, and they were markedly weaker than the left arm and hand, the right fore and upper arm measuring half an inch less in circumference than the left.

There was no apparent affection of the facial muscles on the right side.

The knee, Achilles, triceps, and supinator jerks were all distinctly more active on the right than on the left side. Stroking the sole of the foot gave a distinct flexor response in both feet.

No interference with sensory functions could be demon-

strated. Sensation appeared to be as acute on the left as on the right side of the face, the nævoid condition of the skin not interfering with its sensory functions at all.

No other material facts were elicited during the examination.

Operation.—Chloroform anæsthesia.

Following Cushing's suggestion, the left external carotid artery was first ligatured to check hæmorrhage from a possibly nævoid dura.

An osteoplastic flap was then turned down, exposing the left motor area, with the arm centres about the middle of the exposed area. The bone was not unduly vascular, and the dura was found perfectly normal. On reflecting the dura the soft membranes appeared to be slightly thickened and œdematous, and some perivascular thickening along the larger veins was noted. The exposed convolutions appeared very slightly flattened, and there was a corresponding excess of cerebrospinal fluid. The brain pulsated well, and palpation revealed nothing of note. No trace of abnormal adhesions was seen, and a blunt flat director passed for an inch all round the dural opening, between the dura and brain, met with no resistance. The wound was then closed. Healing took place by first intention, and convalescence was uneventful. No apparent benefit resulted from the operation.

Abstracts

ANATOMY

ON AN ABNORMAL BUNDLE IN THE HUMAN RHOMBENCEPHALON. (Ueber ein abnormes Bündel des menschlichen Rhombencephalon.) K. SCHAFFER, *Neurolog. Centralbl.*, Aug. 16, 1907, p. 738.

(384)

THE abnormal bundles which have been frequently described in the brain-stem may be divided into two classes: those which merely represent variations in the course of the pyramidal tracts, and true abnormal bundles between centres which are not normally connected by myelinated fibres. That which the author describes belongs to the latter class.

It was found in the caudal half of the pons. In the most caudal sections it lay, directed dorso-ventrally, in the fillet layer;

further frontalwards its dorsal end turned into the area of the central tegmental tract, and here it broke up into smaller bundles, which joined the arcuate fibres of the tegmentum of the pons. At the same level its ventral end turned frontalwards and joined the pyramidal tract, in which it could be traced as a definite bundle as far forwards as the trigeminal nucleus.

Although part of it thus ran along with the pyramidal tract, its connection with the arcuate fibres of the pons makes it improbable that it belonged to the pyramidal system.

GORDON HOLMES.

A CONTRIBUTION TO THE LOCALISATION OF THE MOTOR
(385) **CORTEX. (Contribution anatomo-pathologique à l'étude des localisations motrices corticales.)** I. ROSSI and G. ROUSSY, *Rev. neurolog.*, 1907, p. 785.

THIS paper is founded on the study of the histological changes in the nervous system of three cases of amyotrophic lateral sclerosis; the methods of Marchi, Weigert, and Nissl were employed. No reference is necessary to the changes described in the lower motor neurones or in the spinal cord; they were typical of the disease. There was, as in many other published cases, slight degeneration of the dorsal longitudinal bundles in the bulb and in the lower half of the pons; and in the two cases in which it was examined degeneration was revealed by Marchi's method in the middle portion of the corpus callosum.

The cortical changes are of most interest, but it seems that only the post-central and pre-central gyri were examined. In the latter there was extensive degeneration of the radial, and slighter affection of the transverse fibres of the cortex, especially of the supraradial layer; in the post-central gyrus, on the other hand, even by Marchi's method, only a few degenerated radial fibres were seen. The cortical cells of the post-central gyrus were normal in all the cases, but in all those of the pre-central gyrus were affected. In one the affection was practically limited to the Betz cells; in another the large pyramidal cells, in addition, had atrophied and many of them had disappeared; while in the third, in which the degeneration of the cortico-spinal tract was of longer duration and more severe, all layers of the pre-central cortex were affected.

The chief aim of the authors was to correlate these pathological changes with the localisation of function in the cerebral cortex. Assuming that amyotrophic lateral sclerosis is a disease limited to the motor neurones, they point out that the marked contrast between the degree of affection of the pre-central and the post-

central gyrus supports the observations of Sherrington and others that the excitable motor cortex lies in the pre-central gyrus alone. But the fact that in their cases degenerated fibres were revealed by Marchi's method also in the post-central gyrus cannot allow them to assume that no cortico-spinal fibres spring from the cortex of this gyrus. GORDON HOLMES.

ON THE ENTRANCE OF THE OPTIC TRACT INTO THE CORTEX IN MAN. (Ueber den Eintritt der Sehbahn in die Hirnrinde des Menschen.) (386) E. NIESSL v. Mayendorf, *Neurolog. Centralbl.*, Sept. 1, 1907, p. 786.

THIS paper, which is a reply to Tsuchida's criticism of the author's previous communications, contains two points of interest. In the first place, that the central optic tract, in which the fibres which carry retinal impulses to the cortex run, enters the calcarine cortex in compact bundles, a condition which the author regards as characteristic of the projection tracts; and secondly, that this tract has no connection with the first and second occipital gyri, which receive callosal but no projection fibres.
GORDON HOLMES.

PHYSIOLOGY.

THE FUNCTIONS OF THE PYRAMIDAL TRACT. (Über die physiologische Wertung der corticospinalen (Pyramiden-) Bahn.) (387) MAX ROTHMANN, *Arch. f. Anat. u. Phys.*, H. 3 and 4, S. 217.

THE pyramidal tract is not the only path by which, under normal conditions, voluntary impulses in apes are transmitted from the cerebral cortex to the spinal cord. After section of this tract, restitution of movements, not only those which are performed by the limb in conjunction with that of the other side, but also isolated movements, rapidly ensues. This is observable on the day following operation.

The author divided the pyramidal tract in the third cervical segment of the cord. The operation involves destruction also of the rubro-spinal tract, but this does not produce more perceptible disturbance of function than pure section of the pyramidal tract. A spastic condition does not supervene.

The question arises whether restitution of isolated voluntary movements is due to conduction of impulses from the opposite cortex by way of the extra-pyramidal fibres on the injured side of the cord, or whether impulses pass from the cortical region corresponding to the severed tract via the corpus callosum to the

cortical area in the other hemisphere, and thence by the intact pyramidal tract of that side to the cord. With regard to the second alternative, we have no proof that impulses can pass in this way by the corpus callosum. A third possibility is that the movements have their origin in the cortex of the same side as the lesion. It has been shown by H. Munk that, when one motor area is completely extirpated, "secondary" movements become restored in the corresponding extremities, and these must be dependent upon the cortex on the same side as the injured limbs. Such secondary movements are not, however, comparable in efficiency or rapidity of appearance with the movements restored after pyramidal section.

If it be assumed that the impulses pass by the extra-pyramidal tracts of the injured side, there remains the possibility that their so functioning is dependent upon the integrity of the other pyramidal tract which may be supposed to exercise an influence upon the anterior horn cells of the injured side. But experiments are detailed which show that restitution of movements after pyramidal section does not depend upon the other pyramidal tract, since such restitution takes place when the cortical origin of that tract has been extirpated. Further, the restitution is not dependent upon impulses which are transmitted by pyramidal fibres arising from the hemisphere in which the severed pyramidal tract takes origin, passing down the cord without crossing and through the grey matter to the injured side. This is shown by experiments in which, in addition to the extirpation of the arm area in one hemisphere and section of the pyramidal tract on the same side, the cord is also cut in the region of the pyramidal tract of the opposite side. Restitution takes place as before, although all uncrossed fibres must have been severed.

The arm deprived of its pyramidal and rubro-spinal tracts possesses, notwithstanding the destruction of the cortical arm area of the same side, considerable isolated arm and finger movements. "Diachisis," described by Monakow, is not observed, nor is there any lack of definite combined movements. It is scarcely correct to use the terms "restitution" or "substitution" in this regard, since the rapidity with which the movements appear after operation leads to the belief that, under normal conditions, motor impulses from cortex to spinal cord are transmitted through extra-pyramidal fibres lying in the anterior and lateral columns. This extra-pyramidal path is of the greatest importance in relation to the learning of new movements, while the impulses for those which have become fixed by practice pass chiefly by the pyramidal tract.

The results of pyramidal lesions in apes differ from those observed in the human subject, in that there is no spasticity nor

contracture, and little increase of the tendon reflexes, while Babinski's reflex does not appear. It is to be noted, however, that when the internal capsule is destroyed in apes together with the lateral nucleus of the optic thalamus, the symptoms closely correspond to those of hemiplegia in man. Further, the physiological results of lesions in man are modified by age, illness, and impairment of brain nutrition through arterio-sclerosis.

Faradic excitability of the cortical limb areas is not abolished by section of the pyramidal tract, nor when the rubro-spinal bundle is also cut, but it is restricted, three or four weeks after operation, to a circumscribed area of the hand and finger or toe region. This area is in the arm region less in extent after section of the pyramidal tract on both sides than after unilateral section of the pyramidal and rubro-spinal tracts.

The author maintains his belief, in opposition to the views of Grünbaum and Sherrington and others, that the ascending parietal convolution forms part of the motor area. Where the pyramidal and rubro-spinal bundles have been cut, however, the ascending parietal convolution is found to be non-excitable. This is considered to be a pathological result, since under normal conditions this gyrus possesses foci excitable by the faradic current, although these are less in extent and require stronger stimuli than the centres situate in front of the Rolandic fissure.

The author is of opinion that electrical excitability is not a test of whether a cortical area possesses or does not possess motor functions. Although the excitable foci always lie within the motor regions, the latter extend beyond the limits of the former. Further, the boundaries between cortical areas determined by recent histological research are not to be depended upon as indicating the limits of functional areas. The area containing giant pyramidal cells is not to be identified with the electrically excitable region, nor with the motor area of the cortex, nor with the origin of the pyramidal tract. This applies not only to lower apes, but to the anthropoid and man. The ascending parietal convolution is not regarded by the author as exclusively sensory.

W. A. JOLLY.

STIMULATION OF THE PATH OF THE PATELLAR REFLEX.

(388) (**Über Bahnung der Patellarreflexe.**) KRONER, *Neurolog. Centralbl.*, Aug. 1, p. 700.

THIS paper is concerned with the various means of more easily evoking a diminished knee-jerk. Kroner shortly describes first of all the previous methods under two headings: (1) those, such as Jendrassik's, Kroenig's, etc., which aim at diminishing the cerebral inhibition; and (2) those, such as local faradism, that

aim at stimulating the activity of the afferent paths. He then suggests that the motor path may similarly be stimulated to greater activity so that an otherwise absent response may be elicited. Such a method is voluntary contraction of the quadriceps as in walking. He discusses whether this process acts by stimulation of the afferent or efferent paths, and concludes that it acts in both ways. ERNEST JONES.

THE RHYTHMICAL ACTIVITY OF STRIPED MUSCLE. (Über die rhythmische Tätigkeit des quergestreiften Muskels.) (389) SAMOJLOFF, *Arch. f. Anat. u. Phys.*, H. 3 and 4, S. 145.

THE rhythmical activity of the curarised frog's sartorius has been studied by the author under the same conditions as regards temperature, composition of the fluid in which the muscle is immersed, etc., as employed by Biedermann.

The contractions are graphically recorded by photographing upon a moving sensitive surface, and arrangements are also made for stimulating the muscle when required by single induction shocks.

Some of the records of rhythmical movements obtained show a series of simple regular contractions of similar amplitude. Others exhibit more complex forms of curves. These latter curves are to be explained as due to the super-position of contractions initiated from one focus in the muscle upon those beginning at another. In the case of simple contractions, while the successive curves resemble one another closely, the interval between the curves tends to become longer. When a contraction induced by electrical stimulation is interpolated in a series of rhythmical contractions the rhythm is not broken, nor are the subsequent curves affected by the stimulation. This differs from what is observed in the case of the heart, where the systolic rhythm is interrupted by an electrical stimulation, and the systole induced becomes the first member of a new rhythmical series. The author considers that the analogy between the rhythmical activity of heart muscle and striped muscle is not a close one, the latter possessing a rhythm *sui generis*. A closer resemblance to what occurs in striped muscle is to be found in the contractions of the lymph heart of the frog described by Langendorff and Brücke. W. A. JOLLY.

THE RELATION OF THE NERVOUS SYSTEM TO ABSORPTION. (Über die Beziehung des Nervensystems zu den Resorptionsvorgängen.) (390) NOLL, *Arch. f. Anat. u. Phys.*, H. 3 and 4, S. 349.

IN order to determine the relation of the nervous system to processes of absorption, observations were made on the lining epithelium

of the frog's intestine after injection into the lumen of a 2·5 per cent. solution of sodium oleate in distilled water. The injections were made directly into the intestine by means of a syringe and cannula passed, under anæsthesia, through the mouth, œsophagus, and stomach. The animals were killed in some cases six, in others twenty-four, hours after injection, and the intestine treated with osmic acid, frozen and cut.

Comparisons were made between the appearance of the epithelial cells in uninjured frogs and in animals whose central nervous system had been destroyed by pithing before injection. The amount of absorption which had taken place was gauged by the degree in which the cells were stained with osmic acid. As judged by this standard, absorption is less in animals deprived of the central nervous system. Many epithelial cells are unstained, and in those in which reduction of osmic acid has occurred the staining is less intense. The author considers the possibility that the soap solution may be absorbed by the cells equally in both cases, but that in the injured animals the further processes of production of fatty acid and fat within the cells may be hindered. Addition of acetic acid to the osmic acid solution does not, however, increase the staining.

It may be suggested in explanation of the reduced absorption that the contents of the intestine may be affected by the absence of the central nervous system, the digestive secretions being diminished, but it is shown that injection of the intestinal contents of normal frogs during fat absorption into the intestine of the pithed animal does not increase absorption.

The true explanation is to be found in the fact that destruction of the central nervous system impairs the circulation. It is found that when the frog's vessels are perfused with ·8 per cent. solution of sodium chloride, absorption by the intestinal cells of the pithed animal is increased, so that in one experiment the staining with osmic acid was found in places to equal that obtained in the intact frog. As in the course of perfusion the lumen of the gut becomes filled with salt solution, this may exercise its influence on the cells through its presence either in the lumen or in the blood-vessels. The epithelial cells vary among themselves. All are not equally affected by impairment of circulation. W. A. JOLLY.

PATHOLOGY.

(391) THE VARIOUS FORMS OF LACUNAR LESIONS OF THE NERVOUS SYSTEM IN MENTAL DISEASES. (Le varie forme di lesioni lacunari del sistema nervoso nelle malattie mentali.) M. SCIUTI, *Annali di Nevrol.*, Fasc. 1-2, 1907.

THE first form of cavity formation in the central nervous system described by the author is that of lacunæ with disintegration. The lesion consists in the appearance of small cavities, chiefly in the lenticular and caudate nuclei. In each of these cavities a small artery, which is always previous, is found.

The walls of the vessels show evidences of a peri-arteritis, rarely an atheromatous or hyaline condition, although these changes may be present in the vessels of the rest of the brain. The muscular sheath is atrophied ; the adventitial sheath shows proliferation and infiltration. The lymph sheath around the vessel is swollen, infiltrated, and separated from the vessel wall, but not broken.

The wall of the cavity is irregular, and consists chiefly of hypertrophied neuroglial tissues in which rarefaction is taking place.

The nerve fibres and cells have to a large extent disappeared.

This hypertrophic condition of the neuroglia is most intense towards the cavity, and gradually lessens towards the sound tissues.

The other pathological conditions found in these cases are those which are characteristic of arterio-sclerosis and senile dementia. Chronic nephritis is invariably present.

The author attributes an acute toxic origin to these cavities. This acute is followed by a chronic stage, in which the nervous tissues disintegrate, and an irritative infiltration and neuroglial overgrowth results. This inflammatory change is certainly secondary.

The next condition described is that of dilatation of the lymph spaces around the arteries of the basal ganglia. In these cavities there is a small vessel as before, but the wall is smooth, white, and shows no sign of any inflammatory reaction. The nervous tissues around are not altered. This condition is produced by repeated and prolonged stasis of the lymph.

The État criblé of Durand-Fardel is next referred to. This condition is met with generally in the island of Reil and in the tip of the temporal lobe. Rarely it is seen all through the brain.

The small cavities are situated in the centre of the convolutions. They have an artery in the centre which may be normal or may show slight sclerotic changes. The perivascular sheath is

dilated and separated from the wall of the vessel. The nervous tissues are a little rarefied. This condition is also produced by a stasis of the lymph stream.

In other instances there is a dilation of the lymph spaces throughout the central nervous system, especially in epileptics and general paralytics, and in cases of chronic renal disease. A similar condition is found in those who die in status epilepticus. There is some degeneration of nerve tissues, but no sign of inflammation.

The mechanism of the production of this condition is not clear, but it is probable that the disturbance of the blood circulation in the brain reacts on the production, absorption, and circulation of the lymph.

Next, the author mentions the "Stato Cribroso" of Vassale. This condition consists in the formation of small cavities by the degeneration and disappearance of the myeline sheath of the larger nerve fibres.

In the centre of the cavities an altered and often twisted axis-cylinder is found. A similar appearance has been induced in dogs by producing a disturbance of the circulation of the cerebro-spinal fluid.

The condition of État vermoulu is then described. This condition is due to the blocking of capillaries which are already sclerosed. From this there results a breaking down of the nerve tissues, an overgrowth of neuroglia, and the appearance of granular corpuscles.

Lastly, the author mentions the condition of porosis. This is a cadaveric change, in which the cavities are produced by the formation of gas by organisms which have invaded the central nervous system in the last hours of life. Similar cavities have been produced experimentally. R. G. Rows.

PSYCHOLOGY.

A SYMPOSIUM ON THE SUBCONSCIOUS. Morton Prince, (392) Hugo Münsterberg, Ribot, Jastrow, Pierre Janet, *Journal of Abnormal Psychology*, April and June 1907, Vol. ii., pp. 22 and 58 (44 pages).

Dr Prince has had the happy idea of bringing together the views of the above distinguished authorities on this difficult subject. In a prefatory note he first formulates six of the commoner significations of the term. These are: (1) That portion of our field of consciousness which at any given moment is outside the focus of our attention, *i.e.* mental processes of which we are only partially aware; (2) mental processes which are dissociated from the main

personal consciousness, *i.e.* the disaggregated systems characteristic of hysteria; (3) a synthetised body of dissociated processes which constitute a self-conscious ego separate from the conscious self; (4) mental processes of two classes, (*a*) the active dissociated processes of group 2, and (*b*) memories which are either forgotten or at all events out of mind for the moment and are inactive; (5) the main reservoir of mental processes out of which the personal consciousness flows as a subordinate stream; (6) processes which, strictly speaking, are not mental at all but physical; on this view automatic writing and so-called subconscious solution of problems are explained as pure neural processes unaccompanied by any mentation whatever, very much on the lines of the old hypothesis of Carpenter's "unconscious cerebration."

Münsterberg discusses the third, second, and sixth of these views, calling them respectively those of the laymen, the physician, and the theoretic psychologist. He gives a clear exposition of these different points of view, and accepts the third, mainly on philosophical grounds. He sees no reason to conceive a psychical act existing outside of consciousness. Ribot in a short note adopts the same standpoint. Jastrow does not deal explicitly with the mental *v.* physical nature of subconscious functioning, but points out how important it is that any hypothesis of the latter should equally account for both normal and abnormal phenomena. He considers that the evidence is overwhelmingly against a separate and distinct subliminal self, though he recognises the importance that the study of subconscious processes has for both normal psychology and practical therapeutics.

Janet restricts the term subconscious in the second sense given above, *i.e.* to mental systems disaggregated from the main personality. He does not discuss whether forgotten memories in the normal are of a psychological or physiological nature, as he does not think enough is known about the subject. He strongly controverts the view that processes subconscious in his sense have no mental accompaniment, and points out that if we call a somnambulist (using, of course, this term in the French sense, not in the English) an automatic doll, we must logically say the same of every creature.

Morton Prince, in summing up the discussion, brings an imposing array of evidence in support of the view that dissociated activities are really mental. He prefers to call what Janet terms subconscious processes "co-conscious," for the main personality may or may not be aware of them. He finds it more difficult to conceive a physiological process, of a complexity and character capable of exhibiting itself as a spontaneous volitional intelligence, without corresponding correlated ideas, than to conceive such a process with these ideas. ERNEST JONES.

CLINICAL NEUROLOGY.

A CASE OF PRIMARY LATERAL SCLEROSIS. (Ein Fall von primärer systematischer Degeneration der Pyramidenbahnen —Spastische Spinalparalyse.) KATTWINKEL, *D. Zeitschr. f. Nervenheilk.*, Bd. 33, H. 1-2, S. 1.

(393)

THIS appears to be a genuine case of pure pyramidal tract degeneration. Erb's diagnostic quartet of symptoms was present—motor weakness, muscular hypertonus, increased tendon reflexes, and Babinski's sign. The patient was a man of 60, who for a long time had felt his legs weak and stiff, the right being more involved than the left. The weakness next spread to the right arm, and finally the left became involved to a slight extent also. His speech was slightly interfered with, and spasmodic fits of coughing were frequent. Objectively there was considerable wasting of muscle (from disuse, probably), some spastic contractures on the right side, while his gait was of the spastic-paretic type. There was no disturbance of sensibility beyond a slight non-appreciation of heat over the legs, where the skin was persistently blue and cold. The tendon reflexes of both upper and lower limbs were much exaggerated; there was ankle clonus and Babinski's sign. The sphincters were unaffected.

Microscopic examination of the spinal cord showed a degeneration of the crossed pyramidal tract, almost complete in the lower portion of the cord, diminishing in intensity upwards. The right side throughout was more degenerated than the left. In the upper dorsal and cervical regions the direct pyramidal tract also showed some degeneration, especially on the left side. The pyramidal tracts in the medulla were also considerably affected, but above this level they appeared quite healthy. All other parts of the spinal cord seemed normal, as did also the nuclei in the medulla and pons and the motor cortex of the cerebrum. Nothing was made out as to the pathogenesis of the condition.

J. H. HARVEY PIRIE.

MONGOLISM. (Der Mongolismus.) H. VOGT, *Zeitschr. f. d. Erforschung des jugendlichen Schwachsinns*, 1907, Bd. i., H. 6, S. 445.

(394)

THIS is a very valuable review, forty-five pages long, of the whole subject of Mongolism, one that is now arousing considerable though delayed interest in Germany. There is a good account of the literature with a full bibliography, in which ample justice is done to the English work on the subject. Vogt has studied a large number of cases, and analyses in detail the symptoms, course,

etc., in the light of this experience. The whole subject is dealt with so fully that it is impossible to abstract the details. Vogt has used thyroid treatment extensively but says that only those symptoms are improved which are not characteristic of the affection. He opposes Bourneville's view that an anomaly of the thyroid secretion is at the basis of the condition.

ERNEST JONES.

BLADDER TROUBLES OF CEREBRAL ORIGIN. (Über cerebrale (395) Blasenstörungen.) MIECZYSLAW MINKOWSKI, *Deutsche Zeitschr. f. Nervenheilk.*, Bd. xxxiii., S. 127.

THIS is a valuable review, thirty-three pages long, containing brief accounts of nineteen cases and an exceedingly valuable list of references. The author first considers fully the anatomy and physiology of the nervous connections of the bladder. He accepts Von Zeissl's view that the internal sphincter is far more important than the external, and lays stress on the independence of sphincter relaxation and detrusor contraction; the former action may be affected apart from the latter. Discussing the question of the lumbar or sacral bladder centre he thinks that L. R. Müller's researches demonstrate the non-existence of this separate centre, apart of course from the existence of a junction between the upper and lower efferent neurone systems. He accepts the importance of Bechterew's work concerning the cerebral localisation of bladder regulation in the optic thalamus, and perhaps also corpus striatum.

On the clinical side he fully supports E. D. Müller's contention that the current view is incorrect which maintains that bladder disturbances occur in cerebral lesions only when these are bilateral. Like this writer he finds slight bladder disturbances to be very frequent in ordinary hemiplegia, and reports nineteen such cases in support of this contention, as well as referring to numerous others in the literature. Vesical tenesmus is the commonest symptom and often the only one; difficulty in micturition is almost as common. The latter symptom is easily explicable through the obstruction to efferent impulses caused by the lesion. The former symptom he explains by assuming that tonus-inhibiting as well as tonus-increasing impulses reach the sphincter from the cerebrum, so that either incontinence or retention may result according to which set of impulses are most interfered with.

ERNEST JONES.

THREE CASES OF INFECTIVE SINUS THROMBOSIS. J. D. (396) RICHARDS (New York), *Arch. of Otology*, Vol. xxxvi., No. 4.

CASE 1. Female, age not given, a week after the commencement of an attack of acute otitis media developed rigors; at an operation two days later the bulb was found to contain a clot which was dislodged after tying the jugular vein in the neck; the glands over the vein were enlarged. Recovery was uneventful. Author concludes that it is wiser to open the sinus before tying the jugular vein for fear of dislodging a clot by backward pressure.

CASE 2. A middle-aged woman was operated on five weeks after an attack of acute mastoiditis with labyrinthine symptoms. Three weeks later she was seized with a chill, and the sinus was exposed and contained a clot. The jugular vein was also exposed in the neck, and was clotted throughout its length. The patient died of general sepsis.

In CASE 3 a child, æt. 11, was operated on for symptoms of inflammation of the mastoid process, the sinus being involved. Horizontal nystagmus was present and increased on deviation to the diseased side. This symptom disappeared immediately after the operation.

W. G. PORTER.

RESEARCHES ON THE EXISTENCE OF CHOLINE IN THE BLOOD. (**Recherches sur la presence de la choline dans le sang.**) CLAUDE and BLANCHETIÈRE, *Journ. de Physiol. et de Pathol. Gén.*, Jan. 1907, p. 87. (397)

THE authors have undertaken an elaborate physical and chemical study of choline and the methods of its identification. Clinically they employed Donath's method as modified by Wilson, examining sixteen cases of nervous disease in man, and working also with the blood of horses, oxen, sheep, dogs and pigs. In addition they controlled results by seven other modified methods. A full description of the technique is supplied. Their conclusions are:—

1. The body extracted by Mott and Halliburton, afterwards by Donath, is really chloroplatinate of choline, and not chloroplatinate of ammonium or potassium.
2. Choline is not normally present in blood serum.
3. It appears in all probability as the result of the decomposition of lecithine.

S. A. K. WILSON.

MYOTONIA ATROPHICA. W. FÜRNROHR, *D. Zeitschr. f. Nervenheilk.*, Bd. 33, H. 1-2, S. 25. (398)

IN an interesting paper the author describes two cases of this disease, which may be looked upon as a variety of the better known

myotonia congenita. The first case was that of a man of 33, who for some years had observed stiffness in his gait; his hands had also been slightly stiff in their movements for many years. The muscles of the hands gave the characteristic electrical and mechanical myotonic reactions. There was in addition considerable wasting of the muscles of the forearms, and marked wasting of the upper thigh muscles. Considerable improvement resulted from the use of CO_2 baths, massage, and faradisation. The second case was a man of 50, who for long had suffered from a progressive muscular atrophy, which had gradually involved almost the whole body musculature. He was thought at first to be an atypical case of progressive muscular dystrophy, for there was little suggestive of myotonia, save a slight stiffness when commencing to walk—a stiffness which soon wore off. None of the muscles showed an E.R.D., but many a characteristic myotonic reaction.

From a survey of these and other recorded cases the author concludes that there is a "myotonia sine tonu." The diagnosis rests chiefly on the presence of the myotonic electrical reaction. The myotonia usually precedes the atrophy by a considerable time. Few or many muscles may be involved, and, though there is no apparent preference for any particular groups of muscles, the small muscles of the hand are frequently atrophied (they were spared in his first case). J. H. HARVEY PIRIE.

THE OSTEOHYPERTROPHIC FORM OF TABETIC ARTHRO-
(399) **PATHY. (Forme ostéohypertrophique de l'Arthropathie Tabétique.)** KLIPPEL et MONIER-VINARD, *Rev. Neurol.*, 30 juillet 1907, p. 743.

Two types of tabetic arthropathy are widely recognised—firstly, that form in which there is more or less absorption of the ends of the bones with sooner or later dislocation at the joint, and secondly, the type in which hypertrophy of the synovial fringes, a velvety condition of the cartilage, and intra- and peri-articular hyperostoses occur, changes which recall the alterations found in rheumatoid arthritis. The authors would add to these a third type, which they state is seldom recognised, characterised by a striking degree of hypertrophy of the bones. Some writers hold that arthropathies occurring at the hip-joint in tabes are always of the atrophic type. This is disproved by the case here reported, that of a female tabetic in whom an arthropathy at the hip-joint was associated with great increase in size of the femur. The rapid enlargement in these cases may lead to an erroneous diagnosis of sarcoma of the bone. EDWIN BRAMWELL.

HERPES ZOSTER ASSOCIATED WITH PARALYSIS AND
(400) **AMYOTROPHY. (Le zona associé aux paralysies et aux amyotrophies.)** DOUCET, *Thèse de Paris*, 1906.

DOUCET has collected forty cases of this kind, including two not hitherto published. The paralysis almost always occurs in the neighbourhood of the eruption, and is usually incomplete. Herpes ophthalmicus is the variety which is most frequently followed by paralysis. The facial and oculo-motor nerves are most often affected; palsies of the sixth and fourth nerves are much rarer. Paresis of the upper arm and forearm has followed herpes of that region, and paralysis of the leg has been associated with herpes of the great sciatic. Visceral paralysis, *e.g.* paralysis of the bladder and rectum following zoster, is very rare. Only two such cases have been recorded. In Davidson's case the eruption spread over the tuber ischii, the perineum, left half of scrotum, and penis. Retention and obstinate constipation lasted for three days. In Malherbe's case, in which there was also paralysis of the bladder and rectum, herpes was present over the great trochanter, the head of the fibula, and outer surface of the calf.

Amyotrophy is less often met with in zoster than paralysis. It may be either primary or secondary to the paralysis, in the latter case being comparable to the amyotrophy found in acute anterior poliomyelitis. It occurs only in the limbs, not in the trunk or face.

The pathogeny of herpetic paralysis is somewhat obscure. Doucet rejects the fascinating hypothesis of Landouzy, who, after the elimination of certain zosteroid eruptions, ranks zoster with the acute exanthemata, and regards the paralysis of zoster as comparable to that met with in diphtheria. The most probable hypothesis, according to Doucet, is that of a central lesion of toxic or infective nature affecting the origin of several nerves in the cerebro-spinal axis. According to this view the paralysis should be regarded, not as a complication of zoster, but, like the eruption itself, a symptom of the central lesion. J. D. ROLLESTON.

POLIOMYELITIS FOLLOWING VACCINATION. (Poliomyelitis
(401) **nach Vakzination.)** TRÖMNER, *Neurol. Centralbl.*, Aug. 16, 1907, **p. 778.**

A CHILD, aged 12 months, born of healthy parents, was vaccinated on both arms in August 1906. Constitutional disturbance, manifested by shivering and somnolence, developed during the next two days, and on the fourth, paralysis of the left arm with paresis of the other limb occurred. Gradual improvement took place,

but flaccid paralysis of the left shoulder and elbow persisted, together with slight paresis and atrophy of the right leg. An ætiological connection between vaccinia and poliomyelitis is conceivable, if one regards the latter as being a toxic myelitis of vascular origin and the former as introducing an organised virus into the body. Trömner, however, rejects this hypothesis as improbable, since the prodromal symptoms of poliomyelitis developed several days before the usual time for the generalised infection of vaccinia. J. D. ROLLESTON.

GRASSET AND GAUSSEL'S SIGN OF ORGANIC PARALYSIS OF
(402) **THE LOWER EXTREMITY.** NOICA and COHEN, *Journ. de Neurol.*, June 20, 1907, p. 221.

THE hemiplegic patient lies on his back with arms folded, and on request can raise either leg off the bed separately, but he is unable to lift them both simultaneously.

Obviously the hemiplegia must be incomplete in order to allow elevation of the limb at all, and at the same time the paralysis must be sufficiently marked on certain muscles in order that the elevation be not a normal one. These muscles are, according to Grasset and Gaussel, the posterior spinal muscles, the extensors of the trunk. Their theory is that the paralysis of these muscles prevents the fixation of the pelvis on the trunk, and so the legs cannot be lifted.

The authors corroborate the contention of Grasset and Gaussel, but as they have found the sign present in three old men who showed no indication of organic disease of the nervous system, they question the universality of its application.

S. A. K. WILSON.

A CASE PRESENTING THE SYNDROME OF BENEDICKT. (Un
(403) **cas de Syndrome de Benedickt.**) LÉOPOLD-LÉVI et PÉCHIN, *Rev. Neurol.*, 30 juillet 1907, p. 471.

THE patient, a child aged four years, presented a left hemiparesis, with involvement of the lower part of the face and increase of the knee-jerk on this side, but no ankle clonus and no Babinski sign. In addition, there was tremor of the left arm and leg, at times present in slight degree even when the parts were at rest, but much more marked on movement, partial paralysis of the right third nerve, and a more recent though less pronounced paresis of the third nerve of the left side. These symptoms, which were progressive, had developed after an attack of influenza five months

previously. There had been headache but no vomiting, and the optic discs were normal. The lesion was regarded as a tumour, probably tuberculous. Gilles de la Tourette and Jean Charcot, in two of six cases presenting the syndrome of Benedickt, found some paresis of the third nerve on the same side as the hemiplegia. Lévi and Bonniot have described a case of this syndrome, in which they maintained that the hemiasynergy which was present was to be explained by a lesion of the fibres of the superior cerebellar peduncle. EDWIN BRAMWELL.

(404) **A CASE OF SPINAL CORD COMPRESSION, WITH STUDY OF THE SECONDARY DEGENERATIONS. (Ein Fall von Rückenmarkskompression mit Untersuchung der sekundären Degenerationen.)** JENÖ KOLLARITS, *D. Zeitschr. f. Nervenheilk.*, Bd. 33, H. 1-2, S. 91.

THE compression in this case resulted from a sarcoma of the vertebræ pressing on the cord from 6C—3D segments involving also the nerve roots. Clinically the chief interest lay in the intermittency of some of the symptoms and in the unexplained presence of nystagmus.

In the compressed portion of the cord primary and secondary degeneration were indistinguishable. Schulze's comma tract was well demonstrated at various levels. In the 4th cervical segment it appeared as a thin strip of healthy fibres between the degenerated columns of Burdach and Goll. Below the affected area it was seen as a band of degenerated fibres in the same position; the band ran parallel to the posterior horn, extending almost up to the posterior commissure, but stopping some distance short of the periphery. Descending the cord the comma became shorter, and disappeared about D8.

From D4 to D12 there was another degenerated area in the posterior columns, lying almost close to the middle line (the septo-marginal tract?). In its upper part it appeared as a somewhat long oval (in transverse sections of the cord), often in contact with Schulze's tract just behind the posterior commissure. It increased in size downwards as Schulze's tract diminished, at the same time becoming longer and narrower in shape and extending quite close up to the posterior commissure.

In the cross pyramidal tract the longest fibres suffered most; just below the seat of compression only a few of the fibres were degenerated; in the lumbo-sacral cord practically all were.

J. H. HARVEY PIRIE.

JACKSONIAN EPILEPSY FROM EXTRA-CEREBRAL TUMOUR.

(405) **(Zur Klinik der Jackson'schen Epilepsie infolge extra-cerebraler Tumoren.)** Z. BYCHOWSKI, *D. Zeitschr. f. Nervenheilk.*, Bd. 33, H. 1-2, S. 53.

A MAN of 62 for six years had epileptic attacks, of frequent occurrence but always of the same nature—twitching movements commencing in the neck and spreading first to the left arm, then to the left leg. After the attack there was usually a transitory complete left-sided hemiplegia and, latterly, frequently also urinary retention and subsequent incontinence. Consciousness was retained completely during the seizures. Between the attacks the power of movement on the left side had latterly become considerably impaired. During the hemiplegic periods his condition closely resembled that of a hemiplegic from hæmorrhage, with this important difference, viz. that neither Babinski's nor Oppenheim's phenomena were present, and the abdominal reflexes were retained.

By operation there was found and removed a small, hard endothelioma adherent to the inner surface of the dura mater and pressing over the lower and middle thirds of the Rolandic area, but principally on the convolutions posterior to the fissure. The result was complete cessation of the convulsive seizures, hemiplegia, and bladder disturbances. Movement of the left side of the body was greatly improved, although not completely recovered.

J. H. HARVEY PIRIE.

THE ACTION OF SCOPOLAMINE ON THE NERVOUS SYSTEM

(406) **AND THE MUSCLES.** PARISOT, *Journ. de Physiol. et de Pathol. Gén.*, 1906, p. 825.

IN six cases of paralysis agitans, with and without tremor, the author found improvement, *i.e.* relief of rigidity or cessation of trembling, for as long as twenty-four hours after an injection. This favourable result especially on the tremors had already been noted by Roussy in Pierre Marie's clinique.

Experimentally the author found that, in the case of the frog, the injection of scopolamine produces first of all a sedative effect which is followed by a period of excitability, and this in its turn gives place to paralysis. In this stage spinal reflectivity is abolished, and nerve terminals are paralysed. Direct electrical excitation of the muscle is followed by reaction, but not indirect excitation via the nerve. The muscular contraction resembles that of a fatigued muscle, with a prolonged latent period.

S. A. K. WILSON.

THE OCCURRENCE OF CALF CRAMPS IN EASTERN REGIONS IN OLDEN AND RECENT TIMES. (Das Vorkommen von Wadenkrämpfen im orientalischen Gebiete in alter und neurer Zeit. Mediko-historische und folkloristische Studie.) NÄCKE, *Neurolog. Centralbl.*, Sept. 1, p. 792.

(407)

THIS paper represents a continuation of Näcke's historical investigations on cramp, some of which were abstracted in the *Review of Neurology*, August, p. 639. He has inquired amongst the literary authorities on various countries, China, Japan, Arabia, India, etc., and finds that though cramps and convulsions are recorded from the oldest times in all countries, yet calf cramp was not described until comparatively recently. Some nations, as the Egyptians, had no word for calf, still less for calf cramp.

ERNEST JONES.

SOME CONSIDERATIONS ON EPIDEMIC CEREBRO-SPINAL FEVER. WILLIAM WRIGHT, *Lancet*, Sept. 7, 1907, p. 687.

(408)

THE first authentic case of epidemic cerebro-spinal meningitis in the present Glasgow outbreak occurred in March 1906. The disease was made notifiable in the following August. From March to December, 205 cases came under the notice of the Health Department: 30 per cent. of the cases occurred under two years of age, 55 per cent. from two to fifteen, and only 5 per cent. over twenty-five. In only fourteen instances did two cases occur in one household, and in two instances three cases. No secondary attacks have occurred. The mortality during the above-mentioned period worked out at over 66 per cent. EDWIN BRAMWELL.

EPIDEMIC CEREBRO-SPINAL MENINGITIS. WM. ROBERTSON, *Brit. Med. Journ.*, July 27, 1907.

(409)

AFTER briefly reviewing the historical aspect of the disease, the author recounts the outstanding features of the epidemic which attacked Leith during the winter and early spring months of 1906 and 1907 respectively. In the first place it is significantly pointed out that on the Continent, America, and in this country the chief centres of attack have been sea-port towns, inland districts being subsequently invaded. The disease has, the writer thinks, been conveyed to Scotland by aliens who carried the meningococci in their posterior nares and air passages, the micro-organisms being diffused through the agency of sputum. That the over-sea convey-

ance of infection is highly probable is suggested by the fact that the great proportion of cases of illness in Leith occurred among those whose work was intimately connected with shipping, namely dock labourers. An independent investigation made by J. D. Comrie and J. S. Fraser of Edinburgh (*vide* Abstract, p. 632) revealed the existence of cocci, indistinguishable from those of epidemic cerebro-spinal meningitis. These cocci were recovered from the engine-room of a steamer, on board which five men worked in a gang. Each of these five men conveyed infection home, and cases of epidemic cerebro-spinal meningitis developed among younger members of their families. Meningococci were recovered from the upper air passages of "contacts" whom the author of the present paper terms "intermediaries" or infection carriers.

The writer deals with the varying types of the disease that may be encountered, and remarks upon the great difficulty of prognosis as well as accurate diagnosis. The mortality in the Leith outbreak was 100 per cent. in children under 1 year, 83 per cent. from 1 to 5 years, 65 per cent. between 5 to 15, 57 per cent. between 15 to 25, and 77 per cent. between 25 to 60. The periods of illness varied from a few hours to 200 days, extreme emaciation preceding death in the chronic cases. Treatment was not encouraging, hot baths being the most satisfactory routine. Serum-therapy was not a success. To control the spread of the disease, abundant fresh air and cleanliness are advised. Frequent gargling of the throat is also strongly recommended. Chlorine water was used as a throat and nose medicament. Radical disinfection is also necessary. Dr Robertson's suggestion regarding the spread of infection through the throat is supported by Westenhoeffer, who declared that "the tonsillar affection was the primary one." This suggests the bacteriological examination of throats as is done in diphtheria. WM. ROBERTSON.

A CASE OF SEROUS MENINGO-ENCEPHALITIS WITH AUTOPSY REPORT. (410) ALBERT BLAU GOERLITZ, *Arch. of Otol.*, Vol. xxxvi., No. 4.

A LACK of post-mortem reports on such cases prompts the publication of this case.

A child, æt. 2¾, developed an attack of otitis media acuta in the right ear three weeks after the onset of measles. This was followed by gradual loss of sight and hearing, left-sided ptosis, and occasional convulsions of the left arm and leg. Both optic discs were pale, pupils sluggish. Incontinence of urine and fæces was present. At the operation the dura was found to be tense and injected, and on incision abundant clear fluid was evacuated,

but no pus. At the autopsy—dura moderately injected, convolutions flattened, no evidence of tubercle. The ventricles were enormously dilated, and contained a great deal of watery fluid. Author looks on the case as one of acute internal serous meningitis with encephalitis. W. G. PORTER.

MENINGO-ENCEPHALITIS—CLINICALLY, ACUTE DELIRIUM.

(411) (**Über Meningoencephalitis unter dem klinischen Bilde des Delirium acutum verlaufend.**) R. FINKELNBURG, *D. Zeitschr. f. Nervenheilk.*, Bd. 33, H. 1-2, S. 45.

A BOY of 10, after some days of slight headache, became acutely ill with short syncopal attacks and succeeding choreiform movements, and sudden twisting movements of the trunk, consciousness being retained. Then followed a state of dreamy stupor, passing on to a condition of delirium with marked motor unrest. There were none of the usual signs of meningitis. His temperature rose to 106° before death, which ensued nineteen days after the onset.

Post-mortem.—There was found a lepto-meningitis over both frontal poles and in the neighbourhood of the central convolutions. Microscopically this was seen to be an acute exacerbation of a more chronic meningoencephalitis which was neither syphilitic nor tubercular in its nature. The extension of the inflammatory process to the brain was slight, and changes in the cortex were mainly of considerable standing, although there was some recent acute inflammatory reaction. J. H. HARVEY PIRIE.

OTITIC MENINGITIS. ARNOLD KNAPP (New York), *Arch. of Otol.*,
(412) Vol. xxxvi., No. 4.

THE meninges are first infected in about three-fourths of the cases in the posterior cranial fossa, and in about one-fourth in the middle cranial fossa. Forms of meningitis—1. Serous meningitis characterised by an increase of cerebro-spinal fluid which, on withdrawal, is found to be sterile. Here the infective process stops short of traversing the dura. 2. If the infective process perforates the dura, an encapsuled meningeal abscess or a brain abscess may form, or else the infection may extend throughout the arachnoid cavity—general purulent meningitis.

Lumbar puncture is a great aid in diagnosis. The prognosis of purulent meningitis is bad, but the result is not necessarily fatal. Operation should consist in removing diseased bone, with wide exposure of the meninges and incision of the dura.

W. G. PORTER.

KERNIG'S SIGN APART FROM MENINGITIS. (Du signe de
(413) **Kernig en dehors de méningites.)** JOURDAN, *Thèse de Paris*, 1907.

VARIOUS observers have found Kernig's sign either alone or in combination with other symptoms of meningeal involvement in conditions wherein it was subsequently proved that no meningitis had existed. Thus Netter found it in 11·8 per cent. of 313 cases of typhoid, and others have noted its presence in lobar and broncho-pneumonia, in the gastro-intestinal affections of infancy, in glandular tuberculosis, in lumbago and sciatica, and after rachi-cocainisation. On the other hand, cases of meningitis have been recorded in which the sign was absent. It is of more value as an indication of meningitis if it is bilateral than if it is present on one side only, in which case it is symptomatic of alterations in the fibres of the flexor muscles, such as may occur in sciatica, in cerebral nervous lesions, or, generally speaking, in any affection that may cause trophic muscular troubles. In conclusion, the writer thinks that its presence is only of value when made concurrently with an examination of the cerebro-spinal fluid.

J. D. ROLLESTON.

CONJUGATE DEVIATION OF THE HEAD AND EYES AND
(414) **DISORDERS OF THE ASSOCIATED OCULAR MOVEMENTS.** WEISENBURG, 57th Annual Session, American Medical Association, June 1906.

SIXTEEN cases of conjugate deviation of the head and eyes were examined, and in fifteen of these an autopsy was obtained.

Among fifteen "conclusions" the following may be mentioned:—

"It is probable that the cortical oculomotor, head, and combined head and eye centres are subdivided for lateral as well as for upward and downward movements.

"Lesions in the angular gyrus cause conjugate deviation because of involvement of visual and auditory fibres which lie underneath this area.

"The occurrence of conjugate deviation of the eyes or of the head, or of both, is of no value as a focalising symptom, because it may occur as the result of a lesion in any portion of the cerebrum.

"Convergence and divergence have probably cortical centres in the posterior portion of the second and third frontal convolutions."

S. A. K. WILSON.

ENTOPTIC VISION OF THE RETINAL VESSELS ON WAKING.
(415) **(Vision entoptique des vaisseaux rétiniens le matin au réveil.)**
ED. CLAPARÈDE, *Arch. de Psychologie*, 1907, T. vi., p. 269.

CLAPARÈDE has for some eight years observed in himself the following phenomenon. Provided the day is a bright one he sees at the moment of opening his eyes after sleep (at any time of the day) a superb projection of his retinal vessels on the ceiling. The branches of the vessels, which appear black, are most distinct, and the macula appears as a large black spot; the area of the image is about a metre across. He finds in the literature only two cursory references to any similar phenomenon, there being none even in the complete bibliography of entoptic vision given by Helmholtz. Ordinary entoptic vision is due to displacement, by means of lateral vision under certain circumstances, of the shadow cast normally by the vessels on the retina. The parts of the retina that underlie the vessels are hyperexcitable as compared with the rest of the retina. Normally we do not see the retinal vessels, not because we neglect them, as is often asserted, but because the defective illumination of the underlying retina is compensated for by its greater sensitivity.

In the present instance no such mechanism is at work, for the stimulation is direct, not lateral. Three factors probably operate: (1) The general sensibility of the retina is so increased after sleep that the difference between the shaded and unshaded portions is insufficient to compensate the lesser excitation of the former; (2) at the moment of waking, this lesser excitation of the shaded portions is more marked than at any other time, for the light only gradually illuminates these portions by traversing the vessels and being reflected from the neighbouring retina; (3) possibly there is a dilatation of the retinal vessels on waking, and the constriction following illumination causes a clear broad band to be seen round the image of each vessel. Claparède quotes the evidence and arguments in support of these views. ERNEST JONES.

DEFINITION AND NATURE OF HYSTERIA. SCHNYDER, *Journ. de*
(416) *Neurol.*, Aug. 5, 1907, p. 281.

HYSTERIA is the most primitive and the most ordinary expression of the defects inherent in human mentality. The psychical conditions favourable for the development of hysteria are a fantastic imagination, a mental irrationalism which leads the individual to an inexact idea of reality, to an abnormal expression of his affective states, and finally to a more or less complete change in his personality. The psychical peculiarities, however,

are not associated with one mental type exclusively. Hence hysteria presents itself under different guises according to the ethnological and social conditions of its subjects. Moreover, the tendency to-day is to deny to hysteria the character of a morbid entity in order to make of it a modality of those psychical changes which are known as psychoneuroses. Hysterical states are derived by inappreciable transitions from normal mentality. They are constituted by the exaggeration and perversion of the psychical and psychophysical reactions of the normal individual, in particular the emotional reactions and the manifestations of suggestibility. These manifestations are common phenomena in children: one might speak of the physiological hysteria of childhood. In hysteria we see a regression of mentality towards an infantile type, characterised chiefly by weakness of rational judgment. Hysteria is a disease of evolution: in its pure form it is the disease of youth, both of the individual and the race. Similarly, hysteria is rife among rural populations and the artisan class; one still hears from time to time of epidemics of hysteria in country villages which remind one of the classic tales of the Middle Ages.

Apart from these varieties of evolutive hysteria, we must recognise those which develop on the soil of neuropathic heredity in the shape of degenerative hysteria. Here the psychical changes are often unequal: brilliant intellectual faculties may be united to a defective moral sense. The hysteria of this class is not so pure as that of the other division.

S. A. K. WILSON.

DEFINITION AND NATURE OF HYSTERIA. CLAUDE, *Journ. de* (417) *Neurol.*, Aug. 5, 1907, p. 293.

CLINICAL observation shows us that there exists a category of individuals who from infancy exhibit the symptoms of that state of the nervous system which is called "nervosism." This condition is characterised by a defect of regulation of the reflex processes of organic and psychical functions. In the former case, the condition reveals itself in reactions not proportionate to stimuli, in exaggeration or suppression of physiological phenomena of motility, sensibility, secretion, circulation, etc. From the psychical point of view, nervosism is manifested in exuberance of emotion, in exaggeration of receptivity, etc. On this soil grow the neuroses, and hysteria in particular. Hysteria may be considered as a diathesis taking its origin in this constitutional state of the nervous system, and distinguished by the faculty which the subject acquires of isolating certain perceptions or apperceptions consciously or unconsciously, and allowing them to develop such importance that they exercise a considerable dynamogenic action

on various functions. By suggestion or persuasion it is possible to dispel the trouble or to re-establish the lost function.

Neurasthenia and psychasthenia likewise spring from nervosism, but the former is characterised more particularly by a fatigability of the nervous centres; the latter is the expression of a state of constitutional debility and psychical degeneration which favours the erroneous interpretation of endogenous or exogenous sensory impressions. S. A. K. WILSON.

PRESERVATION OF DEEP SENSIBILITY OF THE FACE AFTER (418) DESTRUCTION OF THE FIFTH NERVE. R. H. IVY and L. W. JOHNSON, *Univ. of Penn. Med. Bull.*, May 1907, p. 35.

AFTER reference to Head's conclusions upon the sensory disturbances produced by section of sensory nerves and to the observations of various writers as to the sensory supply of the face, the authors describe the case of a patient in whom there was complete motor and sensory paralysis of the right fifth nerve produced by a tumour situated in the location of the Gasserian ganglion, the facial nerve being intact. From a study of this case and the facts elicited by previous observers, they conclude that the fifth nerve conveys the fibres of sensibility to light touch, changes in temperature, and pain from the cutaneous distribution commonly accorded to it, that the seventh nerve conveys fibres of deep or pressure sensibility from the muscles which it supplies with motor fibres, and that the muscles of mastication are probably also furnished with fibres of deep sensation by that nerve.

EDWIN BRAMWELL.

AKINESIA ALGERA. (**Über Akinesia algera.**) SCHAIKEWICZ, (419) *Neurolog. Centralbl.*, Aug. 16, p. 741.

SCHAIKEWICZ reviews this subject and relates two interesting cases. His conclusions are as follows:—

1. Akinesia algera is a special syndrome occurring in the course of the general psychoneuroses, and constitutes either a complication or an increase of the underlying affection.

2. The pains have a hallucinatory origin. The limitation of movement arises from a general hypochondriac-depressive mood and a secondary hypochondriac-delusional interpretation.

3. The whole picture is allied to hypochondria. The pain-illusions may become fixed illusions analogous with fixed "forced ideas."

4. Akinesia algera is rarely present alone—his case is the only pure one in the literature—but even then it has its hypochondriac character. It is thus to be counted as a variety of hypochondria.

5. The special character of the affection only emphasises its clinical individuality.

It should be mentioned, however, that Schaikewicz makes out a very poor case for the hypochondriacal nature of the syndrome.

ERNEST JONES.

APROPOS OF TACTILE AGNOSIA. DÉJÉRINE, *Rev. Neurolog.*, (420) Aug. 15, 1907, p. 781.

WHEN a subject whose eyes are closed is unable to recognise, or recognises incompletely, an object in his hand, when in other words he is agnosic for that object, it is obvious that he cannot name it because he does not know what it is. In such a subject, his tactile agnosia is always the result either of a defect in peripheral sensibility or of intellectual impairment.

Egger has recently laboured to show that it is possible to recognise the physical or molecular properties of an object, even to have some conception of the form of the object, and yet to fail to recognise it or to name it. In the present communication Déjérine takes exception to the way in which Egger ignores the significance of the fact that in his cases the circles of Weber had to be considerably enlarged before they were appreciated. That fact alone is sufficient evidence of impairment of cutaneous sensibility, and therefore Egger's claim for the existence of tactile agnosia based on defect of cerebral association is scarcely warranted. Déjérine repeats his views on the inaccuracy of the term tactile aphasia, for one can give a name only to what one recognises, and if a patient cannot name an object in his hand, it is because he does not know what it is; there is therefore no impairment of the function of speech, but of the recognition of objects, *i.e.* a tactile agnosia or a tactile asymboly.

S. A. K. WILSON.

THE CLINICAL SIGNIFICANCE OF ALLOCHIRIA. ERNEST (421) JONES, *Lancet*, Sept. 21, 1907, p. 830.

THIS paper, which was read at the International Congress of Neurology on September 5, is a preliminary communication summarising the results, on the clinical side, of a detailed study that will shortly be published. The symptom described under the name of allochiria refers to two entirely different conditions, which hitherto have not been differentiated. The one, *false* allochiria, is an instance of the localisation defect known as alloæsthesia; the other, *true* allochiria, is one of the three varieties of a disaggregation defect here grouped under the designation dyschiria. Alloæsthesic transference of a sensation to the opposite

side can be distinguished from allochiric transference by seven exact characteristics here detailed. It occurs in various affections, notably in tabes, and may be simulated by certain hallucinatory sensations that arise in some hyperexcitable conditions of the lowest afferent system.

The three distinct varieties of dyschiria, here called achiria, allochiria, and synchiria respectively, are briefly described; there are sensory, motor, and mental manifestations of each variety. A stimulus applied to a dyschiric part evokes a sensation having six characteristic attributes, which the author groups under the term Phrictopathic.

The significance for diagnosis is considered from two points of view. First, some of the numerous fallacies in the observation of dyschiria are mentioned, stress being laid on the resemblance of unilateral achiria to hemiplegia. Secondly, the significance of dyschiria as a symptom is considered. The evidence is decisive in favour of the view that it is a specific disaggregation characteristic of hysteria. It is due to a defect in assimilation of the feeling of "sidedness" (chirognostic feeling). The recognition of the dyschiric process is an important step in the exact psychological diagnosis necessary for the elucidation and treatment of hysteria. AUTHOR'S ABSTRACT.

PSYCHIATRY.

A FURTHER STUDY OF ASSOCIATION NEUROSES. J. E. DONLEY, (422) *Journ. of Abnormal Psychology*, June 1907, Vol. ii., p. 45.

DONLEY, in an interesting paper, extends the considerations on this subject that he published in a former article in the same journal. He first defines what he understands by the term Association Neurosis. This is not a disease entity or even a disease form, but merely the symptom of abnormal association formation which is found in a variety of mental states. He points out the great importance of association in mental processes, sensations, emotions, etc., other than purely intellectual processes. The question as to which associations are to be regarded as normal and which abnormal he decides as follows: Those associations are normal whose existence in our present environment is useful, and whose accompaniment in consciousness is pleasurable; and conversely these associations are abnormal whose existence in our present environment is useless or harmful, and whose accompaniment in consciousness is disagreeable or painful. He shews how erroneous it is to speak of normal and abnormal mental associations in the same sense that we speak of normal and abnormal physical processes, for abnormal associations are "governed" by

the same laws of genesis and of maintenance as normal ones, no new factor entering.

He relates five cases of neurasthenia in which abnormal associations were prominent features. ERNEST JONES.

PATHOLOGICAL PERJURY. (Die pathologische Anschuldigung.)
(423) JOHANNUS BRESLER. Carl Marhold, Halle a S., 1907. Pp. 42. Reprinted from *Jur-psychiatr. Grenzfragen*, Bd. v., H. 8.

IN this paper, read before a medico-legal congress at Lublinitz in March of this year, Dr Bresler, *Oberarzt* at the Lublinitz Provincial Asylum, proposed certain reforms in the stipulations contained in Section 164 of the *Strafgesetzbuch* and Section 56 of the *Strafprozessordnung* relating to false accusation. The present Prussian law was, in Dr Bresler's opinion, too restricted in scope; that is, in precisely the same fashion, as there is everywhere a marked tendency to make the exonerative clauses with regard to criminal responsibility more elastic, and so to extend their protection to cases not certifiably insane, so Dr Bresler, for the protection of accused persons, proposed to extend the application of the clauses excluding the subjects of mental disease from giving legal evidence to the subjects of certain other morbid states, *e.g.* hysteria, chronic alcoholism, "pathological lying," etc., and described numerous cases —such as could be instanced in every other country—where innocent persons had been victimised by hysterical and other persons bringing against them charges of criminal assault. The second change proposed was that paragraph 164 of the *Strafgesetzbuch* under which the promulgator of a false accusation can only be proceeded against if the accusation be made against a definitely named or described person, should be so amended that proceedings could be taken against the utterer of any false accusation, such persons being potential dangers to the public.

R. CUNYNGHAM BROWN.

CLINICAL CONTRIBUTION TO THE PSYCHOSES OF DEGENE-
(424) **RATION. (Klinische Beiträge zur Lehre von den Degenerationspsychosen.)** K. BONHOEFFER (Breslau), 1907. Carl Marhold, Halle a S. Pp. 55. Reprinted from the *Sammlung zwangloser Abhandlungen aus dem Gebiete der Nerven- und Geisteskrankheiten*, Bd. vii., Hft. 6.

IN his opening remarks, Prof. Bonhoeffer draws attention to the fact that, as a result of the influence of Kraepelin's classification, the degenerative psychoses figure less largely in modern psychiatry than formerly, the cases which would have at one time come within this category being now considered cases of manic-depressive in-

sanity or of dementia praecox. Prof. Bonhoeffer is convinced that this tendency has gone too far, and that on a foundation of original degeneracy an independent psychosis may develop, which is quite distinct from either of these, and, further, is neither epileptic nor hysterical in nature. To the illustration of this point, he brings an analysis of clinical material from the Observation Station for criminal lunatics at Breslau, with detailed notes of many interesting cases. These cases are such as are well known to prison medical officers in our country, and offer considerable difficulties in diagnosis and prognosis. They give commonly family histories of insanity, alcoholism, suicide or crime, personal histories of idleness, eccentric, perverse anti-social conduct, and frequent petty delinquencies, and finally undergo incarceration for a long period. On reception into prison they are frequently well endowed mentally, and are considered sound and responsible, but in prison they develop more or less suddenly auditory hallucinations, ideas of persecution, of being watched and "influenced," or of food-poisoning; more rarely of ideas of grandeur and of great inventive powers, and at times apparent apathy, resistiveness, and fixed attitudes. These attacks last for varying periods, and they finally entirely recover, either after certification and removal to asylums, or towards the termination of their period of imprisonment, leaving no retrospective alteration in the content of consciousness. The attacks are thus of episodic sub-acute character, and in the non-imbecile cases have to be distinguished from acute paranoia, manic-depressive insanity, dementia præcox, and simulation. Their differential diagnosis and separation as a distinct class is undertaken by the author, who avoids their designation as imprisonment-psychoses, as similar cases have been described under other conditions by Friedmann. He classifies these cases into three groups —(1) simple paranoid psychoses in degenerates; (2) degenerates of querulant type; and (3) cases where mental liability furnishes, for the author, the essential symptoms of degeneracy. In all of these cases, but particularly in the last group, the marked feature of the psychoses is the susceptibility of the subjects to their surrounding conditions, *i.e.* to the inherent and inevitable circumstances of imprisonment. R. CUNYNGHAM BROWN.

TRAUMA OF THE HEAD AS A CAUSE OF INSANITY.
(425) CHARLES W. BURR, *Journ. of the Amer. Med. Assoc.*, Jan. 190.

IN the Annual Reports of the Lunacy Commissioners for England and Wales, accident or injury appears yearly as cause of insanity in from four to five per cent. of the male admissions, and it is as a rule unquestioned that head-trauma may be an efficient cause of insanity. Prof. Burr's brief paper is therefore of interests

Dr Burr believes that trauma must have a suitable soil, an individual already predisposed, or insanity will not develop; and states that his experience has shown him that the vast majority of men who suffer injury to the head do not become insane, no matter what the character and severity of the injury, unless there be so great a loss of brain substance as to produce dementia from direct absence of brain matter or injury to the sensory speech centres.

Even as a predisposing cause, he has never in his cases found injury to be the sole assignable external cause, with the exception of hemiplegic and traumatic epileptic cases, and even in these cases as a rule other conditions—alcoholism, cerebral atheroma, etc.—existed in combination with the injury. Accepting post-traumatic insanity as invariably occurring in a predisposed individual, Dr Burr inquired into its characteristic types. The usual forms he has met with have been confusion, delusion, simple dementia, post-paralytic dementia, and epileptic insanity, but never melancholia or paranoia. R. CUNYNGHAM BROWN.

STUDIES IN PSYCHOPATHOLOGY. BORIS SIDIS, M.A. Boston: (426) D. C. Heath & Co., 1907. Pp. 73. Reprinted from the *Boston Med. and Surg. Journal* of March 14, 21, and 28, April 4 and 11, 1907.

THIS is a study of what the author terms "recurrent psychomotor states," under which term he includes insistent ideas, imperative concepts, persistent or periodically appearing emotional states, irresistible impulses, and the psychomotor attacks designated "psychic epilepsy." All of these are, he maintains, manifestations of subconscious activities, and as they always recur with the same content of consciousness, and with the same motor reactions, they belong to a type characteristic of the lower forms of animal life, and may be regarded from this point of view as reversions to the lower forms of consciousness. Their sudden onset, uniform manifestations, and overpowering effect on the patient's personal consciousness, all point to the same underlying factor of dissociation of the patient's subconsciousness. The term "recurrent mental states" is used in very comprehensive fashion by the author, who subdivides these states into conceptional, ideomotor, sensory, sensori-motor, and motor, and includes within the sensory states functional states of depression and anxiety; the various phobias; the pains, headaches, and hemicranias of functional psychoses; anæsthesias and hyperæsthesias, and other sensory hysterical stigmata; and under sensori-motor and motor hysterical paraplegias, etc., functional tremors, tics, and psychic epilepsy. All of these conditions, he says, can be referred to a disaggregated

subconsciousness; and, further, refer to a past reality now subconsciously real, that is, they are adjustments to past conditions; in fact, "resurrected moments." Proceeding to the clinical illustration of the above analysis of insistent or obsessional states, the author first describes his method of inducing the hypnoidal state—the drowsy condition between waking consciousness on the one hand, and sleep or the hypnotic state on the other—and then gives the results of hypnoidisation in a series of cases of obsession. In all of these cases he has been able to bring to light, by means of the revival of subconscious memory, the circumstances in which were made many years before, and as a rule in early childhood, the powerful impressions which now, though forgotten in waking life, find their painful expression in the various insistent ideas, fears, or conduct of the subject, such expression being permitted by a psycho-physiological dissociation, which inevitably involves a lowering of the threshold of consciousness. The author's exposition of this part of the matter, and of the greater dynamogenesis to which dissociation leads—as, *e.g.*, in psychic epilepsy—and of their relation to periodicity of function is exceedingly interesting. Finally, from his analysis of abnormal mentality, the author draws some conclusions with regard to mental life in general and the treatment of mental states. He shows how, with the gradual evolutional superimposition of inhibitions and the corresponding elevation of the threshold of consciousness, unused energy becomes stored up, civilisation and education being thus economisers of "psycho-neural force." The treatment of insistent states thus lies in the employment of this reserve of energy, and by means of special qualitative stimuli, in hypnoidal or intermediary states, to so use this reserve as to bring about the re-establishment of disturbed inner relations and dissociations. R. CUNYNGHAM BROWN.

TREATMENT.

HYPNOTISM; ITS NATURE, PRACTICE, AND IMPORTANCE
(427) **FOR THE MEDICAL MAN. (Der Hypnotismus, sein Wesen, seine Handhabung und Bedeutung für den praktischen Arzt.)** G. v. VOSS. Carl Marhold, Halle a S., 1907. Pp. 40. Reprinted from the *Sammlung zwangloser Abhand. aus dem Geb. der Nerv- und Geisteskrankheiten*, Bd. vii., H. 7.

THIS is an unimportant paper on this subject, giving a brief historical account of the development of hypnotic treatment, of the phenomena and nature of suggestion, the author's technical methods, and special hypno-therapy. It contains nothing new either in matter or treatment of the subject, and does not call for extended mention. R. CUNYNGHAM BROWN.

TWO PONTINE ANGLE TUMOURS OPERATED UPON. (Ueber (428) **zwei mit Erfolge operierte Fälle von Geschwulst am Klein brückenwinkel.**) OPPENHEIM and BORCHARDT, *Berlin. klin. Wchnschr.*, Juli 15, 1907, p. 875.

THE first case was that of a man aged 26, who suffered from nervous deafness. There was a history of pain and tinnitus in the right ear two years previously, with gradual loss of hearing power and increasing vertigo. On admission to the hospital a diagnosis was made of an endocranial new growth in the posterior cranial fossa, probably in the right pontine angle. At the operation a tumour the size of an egg was removed, which microscopically exhibited the characters of a fibro-sarcoma, and was of a carcinomatous consistency in some places. Relief of the symptoms resulted. The patient was seen three months later, when the headache, vertigo, difficulty in walking, paræsthesia in the right field of vision and choked discs had all disappeared. The second case reported exhibited general and focal symptoms of cerebral tumour. The radical operation was performed, and a fibroma the size of a walnut removed from the left pontine angle in small pieces. The immediate results were satisfactory, but a week later severe bulbar symptoms appeared, with complete anæsthesia in the left trigeminal region and increase of the motor symptoms. The patient subsequently improved, and when last seen there was no headache, vertigo, or vomiting, and the paralysis of the tongue, dysarthria, and paralysis of deglutition had considerably decreased. Reference is made to a case reported by Poppert, in which the tumour could not be localised, and on opening the dura two walnut-sized growths were removed from the pontine angle. Borchardt has operated upon three of these cases, in all of which the lateral site of the tumour was correctly diagnosed. The operation was performed in two stages. In the first a four-cornered flap of soft tissue extending 4 cm. above the occipital protuberance was made, and the bone removed above the posterior cranial fossa, the dura being opened a few days later and the tumour removed. Of six cases in which tumours have been removed from the pontine angle (observed by Borchardt), three of the patients are living. In one of these, in which the tumour was probably not completely removed, the patient had no troublesome symptoms, the headache had disappeared, and the choked discs had not proceeded to atrophy. His cases show that excellent results can sometimes be obtained from the operation, and he thinks that with more extended experience its prognosis will be improved. The smaller the tumour, and the earlier the diagnosis is made, the more chance there will be of relieving the patient of troublesome symptoms and saving eyesight.

DONALD ARMOUR.

Report of Congress

INTERNATIONAL CONGRESS FOR PSYCHIATRY, NEUROLOGY, PSYCHOLOGY, AND CARE OF THE INSANE.

Amsterdam, September 2-7, 1907.

ANY report other than the official one of a congress of this size, which comprised three independent sections, and at which over a hundred and twenty papers were read, must necessarily be both sketchy and incomplete. It is clear, therefore, that the present account represents little more than the personal impressions of one of the participants, and its obvious deficiencies must be attributed in part to this fact.

At the outset it may be stated in an unqualified manner that the Congress was a thorough-going success and amply fulfilled the purposes for which it was intended. It was perhaps the most brilliant gathering of neurologists that has ever come together, for the most distinguished representatives of the science in every country except England were present. The number of adherents totalled over 700. So successful was the reunion that a permanent international committee has been formed to arrange for further congresses to be held at definite intervals, probably of three years. The site of the next is not yet decided, though England was freely mentioned in this connection.

It was naturally a matter of interest to compare the activity of various nations at the Congress. The members came from the most diverse regions, from Chile and Japan to Bulgaria and Portugal. Of the three languages permitted at the meetings, German was by far the most prominent, though French was more widely used for official purposes; English was hardly spoken. Germans were numerically in the ascendant, and there were as many Americans as English. The so-called "practical" attitude of the English nation towards science is well illustrated by the fact that at the sections concerned with Psychiatry, Neurology, and Psychology, 3·5 per cent. of the papers came from Great Britain, whereas at the section concerned with Nursing of the Insane, 30 per cent. of the papers, nearly ten times as many, did so.

The work of the Congress may be considered under three heads: (1) Addresses read before General Meetings of the whole Congress; (2) Discussions on set subjects opened by chosen speakers: these took place in the separate sections; (3) Individual communications to the various sections. We may consider these three groups in this order.

I. General Addresses.

These were six in number, as Professor Raymond was unable to deliver his. They were as follows. Bechterew (St Petersburg) —"Recherches objectives sur l'activité psychique"; Gaskell (Cambridge)—"On the evolution of the vertebrate central nervous system"; Ziehen (Berlin)—"Methoden der Intelligenzprüfung"; Pick (Prague)—"Die umschriebene senile Hirnatrophie als Gegenstand klinischer und anatomischer Forschung"; Van Gehuchten (Louvain)—"Le mécanisme des mouvements réflexes"; MacDonald (New York)—"Development of the modern care and treatment of the insane as illustrated by the New York State Hospital System." Perhaps the two addresses that aroused most interest were those of Gaskell and Van Gehuchten, though there was little actually new in either, as they were summaries of the authors' previous writings in the *Journal of Anatomy and Physiology* and the *Névraxe* respectively. Dr Gaskell illustrated his address with a lantern demonstration of slides which supported his well-known hypothesis that the cerebro-spinal ventricular system was originally an alimentary one. He quoted numerous analogies between the central nervous system of vertebrates and invertebrates, and shewed how the one must have developed directly out of the other. According to his hypothesis the infundibulum represents the old œsophagus, the supra-infundibular brain mass the supra-œsophageal ganglia, the crura cerebri the œsophageal commissures, the infra-infundibular brain mass the infra-œsophageal ganglia, the spinal cord the ventral chain of ganglia, the ventricular cavities the cephalic stomach, and the central canal of the spinal cord—which in the embryo terminates by way of the neurenteric canal into the anus—the straight intestine of the invertebrate.

II. Discussions on Set Subjects.

These, as also the individual communications, must be considered in connection with three sections separately.

A. *Psychiatry and Neurology.*—Nine subjects were selected for discussion in this section.

1. Labyrinth-Tonus. Papers were read by Ewald (Strassburg) and Winkler (Amsterdam). Ewald explains the many distant symptoms—weakness of many muscles, particularly those of the larynx and jaw—that follow labyrinth operations by assuming that the labyrinth exerts a constant influence (labyrinth-tonus) over the homolateral voluntary muscles; removal of this influence leads not to paralysis but to inexactness in movement. He points out that the function of equilibration is more ancient than that of hearing, and tends to diminish in importance as the latter increases, a fact that explains the different results obtained by experimenta-

tion on different animals. Winkler demonstrated in some rabbits the results—immediate and remote—of various labyrinthine operations, and made a most valuable contribution to the anatomy of the central connections of the eighth nerve, tracing in detail the course of the afferent and efferent impulses in relation to hearing and equilibration. The papers were criticised by Van Gehuchten on both clinical and anatomical grounds.

2. Cerebellar-Tonus. A paper was read by Van Rijnberk (Rome) and discussed by Mann (Breslau).

3. Chronic Alcoholic Psychoses. Papers were read by Mott (London) and Schröder (Breslau). Dr Mott's important paper summarised much of his recent publications in the *Archives of Neurology*. He compared the results of 1200 consecutive autopsies performed at Charing Cross Hospital with those of a similar number performed at Claybury Asylum; in the former series there were 82 cases of advanced cirrhosis of the liver, in the latter only one doubtful case. The statistics as to the importance of alcohol in the causation of insanity were often fallacious on account of the varying standard adopted as regards what constitutes an alcoholic history. Dr Mott considers that alcohol is an unimportant cause of insanity, and that in alcoholic psychoses it is the special underlying temperament that is the significant factor. He shewed slides indicating that greater cortical changes were found in Korsakoff's psychosis and delirium tremens than in the so-called chronic alcoholic psychoses—hallucinatory insanity, etc. The condition of acute hallucinosis he had seen most often in individuals who had recently taken to drink. Schröder was similarly very sceptical about much of the current teaching on the importance of alcohol in this respect. He discussed many of the difficulties of the subject, and showed how a closer definition of general paralysis on histological grounds had, in separating from it the alcoholic pseudo-paralysis, diminished the significance of alcohol in respect to this disease.

4. Differential Diagnosis between Dementia Paralytica and other forms of Acquired Dementia. Dupré (Paris) gave a masterly account of this subject, confining himself entirely to the actual psychological characteristics of the various dementias without dealing with the associated mental or physical manifestations; his paper was discussed by Binswanger (Jena), Putnam (Buffalo), and A. Marie (Paris).

5. Modern Theories on the Origin of Hysteria. Papers were read by Pierre Janet (Paris), Aschaffenburg (Cologne), Jung (Zurich), Jelgersma (Leiden). Professor Janet, in a most persuasive address, pointed out that, although the mental nature of hysteria had been widely known for thirty years, even yet the mental manifestations are rarely given that exact study that their

importance demanded. He pleaded that the various hysterical symptoms, even the simplest, should have their psychological characters defined with the same scientific precision that is applied to various physical signs of organic disease. He took hysterical delirium as an example, and showed how its characteristics could be differentiated from those of other deliria. Janet considers it premature to discuss the theory of hysteria until more accurate work has been done on the psychological lines indicated. Aschaffenburg occupied himself mainly with an adverse criticism of the sexual side of Freud's hypotheses. Jung gave a striking historical account of Freud's hypothesis, and traced the development of Freud's views; he quoted some of the evidence on which these were based. Jelgersma described hysterical stigmata in terms of shortened association paths, whereby the mental life is simplified; he considers defective hereditary development as the important basis of hysteria. A heated discussion followed in which Sachs (Breslau), Alt (Uchtspringe), O. Gross (Graz), Heilbronner (Utrecht), Sollier (Paris), Marie (Paris), and Dupré (Paris), took part. It centred round the sexual part of Freud's hypothesis; all those who had had experience of psycho-analysis agreed with the truth of Freud's views; all those with no such experience thought that psycho-analysis should not be undertaken for fear of encouraging undue intimacy between doctor and patient.

6. Actual State of the Anatomical Division of the Cortex. Paper read by Vogt (Berlin).

7. Asymboly, Apraxia, and Aphasia. Papers were read by Pick (Prague), Von Monakow (Zurich), H. Liepmann (Berlin), and Hartmann (Graz). Pick discussed the different senses in which the term asymboly was employed, the three main significations (Kant's, Finkelnburg's, and Wernicke's) being fundamentally different, and urged the necessity of definition of these and allied terms. He suggested that asymboly should signify, as was intended by Finkelnburg when he invented the word, "disturbances of the various means of expression, including speech"; only some of the aphasias are thus included, and asymboly is bounded—though not sharply—on the one side by the agnosias, on the other by the apraxias. Von Monakow used the word Asemia to include two main groups: (1) the aphasias—disturbances of the use and comprehension of speech; and (2) the asymbolies (in Wernicke's sense), divided into sensory asymboly (agnosia) and motor asymboly (apraxia). He gave a demonstration of diagrams of all the published cases in which a post-mortem examination had shewn the presence of a lesion in Broca's area. These cases comprised 14 negative cases, with no symptoms, and 38 positive ones; the latter were 8 chronic cases without improvement, 2 chronic ones

with improvement, 10 acute ones, 13 acute ones with restitution, and 5 of "pure subcortical aphasia." On the basis of these cases he expounded his Diaschisis principle (see *Review of Neurology*, Feb. 1907, p. 151). He thinks that many of the negative cases may have shewn aphasia symptoms for only an hour or so, and considers that many of the problems of aphasia would be solved if attention was given to the differentiating between temporary and residual symptoms. Hartmann defined exactly a number of problems that should be studied, and Liepmann also reviewed the whole field in a broad manner. The subject was further discussed by Heilbronner (Utrecht).

8. Focal Symptoms in Genuine Epilepsy. Binswanger (Jena) read a paper on this subject. He began by a definition of idiopathic epilepsy as a psychoneurosis, various symptomatic epilepsies which he enumerated being excluded. He considers that the post-mortem changes described by Alzheimer and others are not at the basis of the condition, but are *secondary* to repeated attacks which are followed by dementia. He gave a classification and description of the focal manifestations of genuine epilepsy, and laid stress on the very great difficulty of diagnosis between the condition and certain organic cases, particularly those due to slight infantile encephalitis. Heilbronner (Utrecht) and Muskens (Amsterdam) discussed the paper.

9. Secondary Contractures in Hemiplegia. L. Mann (Breslau) read a comprehensive paper on this subject. The observation, first made by Van Gehuchten, that secondary hypertonia selects those group muscles which recover their function, and that paralysed muscles are atonic, has been fully confirmed by later writers. Förster has further shewn that muscles may become hypertonic not only by their recovered capacity allowing them actively to shorten, but by passive shortening, whether this is accidental (in bed) or experimental; Mann thinks that this latter process is of no great importance, though its occurrence to a certain degree is unquestionable. Van Gehuchten's inference from the above facts that the contracture is due to a disproportionate innervation of the less paralysed muscles, and is essentially of the same origin in peripheral and hemiplegic cases, is contradicted by Mann on the grounds that: (1) the contracture is not proportional to the difference in the intensity of paralysis of two antagonistic groups; it is often little marked, particularly in peripheral cases, when one group is completely paralysed, and its antagonist quite spared; (2) the characters of peripheral and hemiplegic contractures are different, the latter being far more variable than the former, and more readily affected by various influences; and (3) the contracture in hemiplegia not rarely precedes by some days the restitution of capacity. He expounded his previously published view that

hemiplegic contracture is due to the fact that the paralysed antagonists cannot be "denervated," as normally they are when a given group is innervated, so that the contrast between the two groups is doubled in intensity; this is based on the belief that innervating and denervating impulses follow the same paths, so that paralysis of a given group implies abolition of its power, not only to contract, but also to relax at an appropriate moment. The paper was discussed by Van Gehuchten (Louvain), who pointed out recent modifications in his hypothesis, Lewandowsky (Berlin), and Van Valkenburg (Bloemendaal). Of the above discussions, the fifth (on Hysteria) and the seventh (on Asymboly and Aphasia) aroused the greatest interest. Perhaps the most striking individual contributions were those of Mott, Dupré, Janet, Jung, and Von Monakow.

B. *Psychology and Psycho-Physics.*—There were five subjects selected for discussion in this section.

1. Actual State of the Lange-James Theory of the Emotions. Papers were read on this subject by Paul Sollier (Paris) and De Sarlo (Florence). Professor Sollier gave an interesting account of the views he has expounded in his recent book on the Emotions. He first ably described the various objections that have been formulated against the theory, which he considers now has merely a historical interest. He grouped the facts that demonstrate its falsity into pathological, psycho-physiological, and physiological. He rejects even the recent ingenious modifications that D'Allonnes has offered of the theory. He then propounded his own position, which is partly based on the belief in intra-cerebral vaso-dilator nerves. He holds that emotion is a manifestation of cerebral æsthesia, not of peripheral sensibility, and is the "feeling of the diffusion of the discharge of cerebral energy." The general attitude towards Sollier's hypothesis was that at bottom it was just as "peripheral" as the Lange-James one, in that according to it emotion is *secondary* to organic changes, and not merely concomitant with these.

2. Psychology of Puberty. Marro (Turin) read a long paper on this subject. The first part of his paper, which contained little that was novel, was mainly a descriptive account of the psychological changes that accompany puberty in the two sexes; in the second part he dealt with the psycho-pathology of the state, laying special stress on the relation of such affections to criminal impulses. He still adopts the "degenerative" view of such affections that is so popular in his country.

3. Difference between Perception and Conception. The two speakers invited were Jodl (Vienna) and Mercier (London). Professor Jodl pointed out, after defining the terms, the conflict between the philosophical and practical psychological attitude

towards this subject. Philosophically it was impossible to make a sharp distinction between perception and conception. This was similarly impossible from the standpoint of individual-psychology, but was a distinction of great importance from that of social psychology, since it was the main criterion of mental unsoundness. He shewed how conception is a process that has no significance except as a social phenomenon.

4. The Secondary Function. O. Gross (Graz) read a very stimulating and broadly conceived paper on this most important subject. It is impossible to give any abstract of his novel views on it, and the reader is referred to Gross's interesting book.

5. The Previous History of Psychopaths. C. Neisser read a paper on this subject.

The first subject was the one that aroused most general interest. Perhaps the most important contributions were those of Jodl and Gross.

C. *Nursing of the Insane.*—Six subjects were chosen for discussion in this section.

1. The Instruction of Nurses, their Rights and Duties. Papers were read by G. M. Robertson (Larbert) and Van Deventer (Amsterdam).

2. The Care of Lunatics who have been brought to Courts of Justice. Papers were read by Morel (Mons) and Van Hamel (Amsterdam).

3. The Organisation of the Administration of Asylums, and State Supervision of Care of the Insane. Ruysch (Hague) read a paper on this subject.

4. The Boarding-out of the Insane in Private Dwellings and in the Country. Papers were read by A. Marie (Paris), Alt (Uchtspringe), and Meens (Gheel). A special conference was also held on this subject.

5. The Education of Backward Children. Papers were read by Ferrari (Bologna) and Trüper (Jena).

6. The Different Forms of Psychotherapy. Van Renterghem (Amsterdam) read a paper on this subject, replying to the criticisms of hypnotism offered by Dubois and Dejerine. He published his paper in the form of a booklet containing the account of many cases. Van Renterghem conducted several parties over his institute for psychotherapeutics, and gave a demonstration of his measures.

III. Individual Contributions.

It is impossible to do more than mention most of these.

A. *Psychiatry and Neurology.* — Three papers from Great Britain were read before this section. Dr Mott read one entitled

"Experimental and Histological Examination of the Cortex of the Lemur's Brain, and a Comparison with that of the Primates in Relation to the Functional and Structural Evolution of the Convolutional Pattern." This comparative study was illustrated by drawings and lantern slides. Drs Orr and Rows gave a lantern demonstration of "Lesions of the Spinal and Cranial Nerves experimentally produced by Toxins." Much of the interesting work on which this paper was based will be familiar to readers of the *Review of Neurology* (see October 1903 and January 1905). More recently the authors have performed numerous experiments in which celloidin capsules of various cultures (staphylococcus, bacillus coli, Gaertner's bacillus, etc.), the virulence of which had been artificially heightened, were inserted in apposition into certain nerves. They have shown that (1) toxins readily spread up spinal and cranial nerves to the central nervous system; (2) in their extra-medullary portion these nerves are protected from the influence of the toxins by the vital action of the neurilemma sheath, but on losing this in their intra-medullary part they at once undergo degeneration; (3) the first change is a primary degeneration of the myelin, the axis-cylinders and nerve cells are evidently affected later. A paper was read on "The Clinical Significance of Allochiria" by the present writer; it is abstracted in the October number of the *Review of Neurology*.

Muskens (Amsterdam) read in English a paper on "Cerebellar Connections." It summarised numerous experimental researches, accounts of which the author has published in the *Royal Academies of Sciences Transactions, Amsterdam*. These were carried out especially from a comparative standpoint, and it is interesting to note that the conclusions thus arrived at coincide throughout with those of Clarke and Horsley. For instance, in rabbits, extirpation of the flocculus led in all cases to degeneration of the ventral cerebello-thalamic bundle of Probst on the opposite side; in squirrels it leads to no degeneration in the superior cerebellar peduncle. In rabbits a part of the dentate nucleus is contained in the flocculus, but not in squirrels. In neither case was any degeneration found in the rectiform body or spinal cord. These facts therefore support Horsley's conclusions that for every connection of nerve fibres issuing from Purkinje cells the basal nuclei are interposed. Similar results were obtained in cats.

De Lange (Amsterdam) read a paper entitled, "Sur l'anatomie du faisceau longitudinal postérieur." He first gave a general account of previous researches and views, and then stated a series of conclusions based on personal researches made on different animals. The chief of these conclusions were as follows: The principal fibres of the bundle are descending ones which go from Darkewitsch's nucleus to the anterior column of the spinal cord;

they are, for the most part, uncrossed. Ascending fibres pass from near the anterior median fissure of the spinal cord to the cranial motor nuclei. From the vestibular nerve fibres pass to all the cranial motor nuclei, more going to the opposite side than to the same side; the same is true of the cochlear nerve. A great number of fibres run from Deiter's nucleus or the neighbouring grey matter to the motor nuclei of either side. There is no direct connection between the trigeminal nerve and the posterior longitudinal bundle.

Lewandowsky (Berlin) read an interesting paper on "Abspaltung des Farbensinnes durch Herderkrankung des Gehirns." This was an account of a case of hemiplegia and hemianopia in which the following condition obtained in respect to the still active field of vision. The patient could not name a given colour shewn him, nor state the colour of a given object; neither could he pick out a colour the name of which was mentioned, or match the colour of a given object shewn him. In spite of this, his colour-sense shewed no defect when tested by the most rigorous methods; that is, he could detect all the differences between colours shewn him. Thus the colour-sense was retained, but it was dissociated from the names of colour and from the perception of objects. We have no knowledge of such a fine dissociation in function apart from hysteria, and it is a pity that Lewandowsky did not state the grounds on which he excluded the presence of this affection.

Forster (Berlin) read a paper entitled "Über die Bedeutung des Affektes bei der Paranoia." This represents an important step in the direction towards which modern psychology seems to be tending, namely, that feeling plays, as maintained by Margulies and Specht, a fundamental part in the origin of paranoia, even in Kraepelin's pure paranoia. Forster freely grants that the separation between intellectual and affective psychoses is largely artificial. He suggests a compromise between the old and the new schools in the view that, though the actual primary defect in paranoia is an intellectual one, it relates only to experiences heavily charged with feeling, which thus is of fundamental significance in this connection. This of course reduces the discussion largely to a matter of words, for if, as Forster seems to maintain, the intellectual defect in assimilation of a given experience is due to the strong affective element of the latter, it is the affection which is of prime importance.

Bychowski (Warsaw) read a paper on "Organische Hemiplegieen ohne Babinski." He has studied a number of such cases that have come to autopsy, and found that they are characterised by the presence of a cortical or meningeal lesion; the same applies to the occasional retention of the abdominal reflex in hemiplegia. He concludes that the presence of an abdominal reflex, or the

absence of Babinski's sign in organic hemiplegia, is good evidence that the lesion is not deep, and that Babinski's sign is valuable in relation only to affections of the pyramidal fibres.

Joire (Paris) discussed the Nature and Treatment of Hysteria. He regards hysteria as a functional disturbance of equilibrium of the nervous system, of such a sort that an excess of energy in one direction (*e.g.* contracture) is counterbalanced by a loss in another (*e.g.* paralysis). He applied this compensation principle to various manifestations of hysteria. Terrien (Nantes) made two contributions, the principal thesis of which was that hysteria is an hereditary and incurable condition, the various accidents of which are evoked by suggestion. Orszanski (Charkow) read a paper on "Die Genese und die Natur der Hysterie," in which he dwelt on hysteria as a reflex disturbance due to manifold organic disturbance; he regards the psychical manifestations as only one part of hysteria.

Louise Roubinovitch (New York) read a note on "Pression sanguine générale et cérébrale dans l'épilepsie électrique." She produced this condition in animals by passing through them an interrupted current of moderate voltage for four seconds. The blood pressure was found to rise from the beginning of the clonic stage, reaching its maximum at the time when the clonic movements were at their greatest. She also gave a demonstration of the resuscitation of electrocuted animals, with a description of the practical application of the method to industrial circumstances.

Indemans (Maastricht) read a paper entitled, "La myasthenia gravis pseudoparalytica comme expression de l'insuffisance de la sécretion interne." He cited a number of arguments, all of which could easily be otherwise interpreted, that had led him to suppose that myasthenia gravis was due to a deficiency of the thyroid, pituitary, ovarian, and testicular secretions.

Among the other papers read before this section should especially be mentioned the following: Oppenheim (Berlin), "Demonstration eines extirpirten Rückenmarkshauttumor." J. W. Putnam (Buffalo), "A comparative clinical study; four cases of Landry's paralysis; two cases of infection of multiple neuritis; one case of myasthenia gravis." Ramon y Cajal, "La dégénération traumatique des cylindres-axes du cerveau et du cervelet" (with demonstration). Jelgersma (Leyden), "Demonstration von Schnitten durch das ganze Gehirn." Wertheim Salomonson (Amsterdam), "Demonstration de l'enrégistrement de l'Électrodiagramme humain à l'aide du Galvanomètre à corde d'Einthoven." Moreira (Rio de Janeiro), "Les maladies mentales du Brésil." Marcus (Stockholm), "Acute Verwirtheitszustände auf syphilitischen Grund." Catzaras (Athens), "Valeur diagnostique et prognostique des symptomes catatoniques." Sollier (Paris), "Un cas de myasthénie hystérique." Dubois (Berne), "Considérations sur le

traitement des phobies." De Vries (Amsterdam), "Démonstration de microphotographie colorée." Gutzmann (Berlin), "Zur Übungsbehandlung der Aphonie," and also "Die Therapie des Stotterns." Marinesco and Parhon (Bucharest), "Recherches anatomo-cliniques sur l'origine du sympathique cervical." Chartier (Paris), "Encéphalite hémorrhagique expérimentale." Faure (La Malon), "La mobilisation methodique et les contractures secondaires des hémiplégiques."

B. *Psychology and Psycho-Physics.*—Lechner (Kolozsvar) read a paper entitled "Ueber negative Sinnestäuschungen." Regarding sense-perception as a complex of reflex processes, he shewed how changes in the arrangement of these processes, partly through increase, partly through deficit of the reflex components, lead to sense deception; those evoked by increase are illusions (exogenous stimulation) and hallucinations (endogenous stimulation). He described the different varieties of negative illusions, which he defined as illusions with manifestations of deficiency (Ausfallerscheinungen).

Bychowski read a paper on "Reflexstudien," which summarises the results of investigations of certain reflexes in infants, undertaken for the purpose of studying which were phylogenetically the most ancient. He found that the patellar reflex was constantly present from the first, that the Achilles reflex appeared considerably later, and the abdominal reflexes much later still. He concludes on these and other grounds that the latter two are probably of cerebral origin, whereas the patellar reflex is purely spinal.

Van Römer (Dutch Navy) read an interesting paper entitled "Über das Verhältniss zwischen Mondalter und Sexualität." Referring first to Perry-Coste's work on sexual periodicity in men, he then gave the results of four years' observation of sexual manifestations in a man. The curves showed an astonishing correspondence with those of Perry-Coste. In each month were two maxima, which coincided with the times of the new and full moon; and two minima, which coincided with the times of the first and last quarters. Whether the relation to the time of the moon will be borne out by further observation is of course questionable, but the present observations are important evidence of some periodicity in the sexual manifestations in men. Van Römer also read a paper on "Eine neue Theorie zur Erklärung psychologischer Probleme," which was mainly an exposition and development of Van Albada's oscillation hypothesis.

Wijnaendts Francken (Hague) read a paper on "Statistisch-sexuelle Traumdifferenzen," based on an inquiry into the dreams of more than 300 educated people. Half of the men dreamt every

or nearly every night, and three-quarters of the women. Sense-perceptions (colour, sounds, etc.) were considerably more frequent in women, as were emotional features; dreams in men were more often colourless in both respects. Men more rapidly and more often forget their dreams than do women.

R. Novoa (Santiago) read a paper on "Temps réflexe et temps consciente," concerned mainly with the psychical aspects of spinal action.

Amongst other papers may be mentioned those of Gutzmann (Berlin), "Über Hören und Verstehen"; Michotte (Louvain), "Sur les oscillations de l'attention"; Sleeswijk (Bloemendaal), "Über die Bedeutung des psychologischen Denkens in der Medizin"; C. Charpentier (Paris), "Un cas de simulation de Jolie—Essai de psychologie criminelle"; Van Wayenburg (Amsterdam), "Le temps de réaction simple appliqué à l'étude des mouvements coordinés"; Joire (Paris), "Sur une force nerveuse extériorée—Présentation d'un appareil sthénomètre servant au diagnostic des maladies nerveuses."

C. *Nursing of the Insane.*—A paper by Easterbrook (Ayr) on "The Sanatorium Treatment of Active Insanity by Rest in Bed in the Open Air" was read in his absence. The reasons why active exercise treatment of insanity had been replaced by rest in bed treatment were fully considered. An account was then given of the experience of open-air treatment—which was warmly advocated—at Ayr Asylum, and the rational grounds for the procedure. Mabon (New York) also read a paper on the same subject, and dealt with the different classes of cases—both acute and chronic—which were most suitable for the treatment.

W. W. Ireland (Musselburgh) read an interesting paper on the "Increase of Diseases of the Nervous System and of Insanity." It contained a collection of statistics which went to shew that this increase was a real one; this seemed to be so, not only in Great Britain, but in practically all civilised countries. The causes of this in the changed conditions of modern life were gone into.

Clay Shaw (London) read a paper entitled "A Contribution to the Analysis of the Mental Process in Criminal Acts." He pointed out that in criminal acts no fresh element enters into the constitution of the mental processes; otherwise expressed, a criminal act is a normal response to an abnormal stimulus or motive; the question is therefore largely, if not entirely, one of correct training.

Toutychkine (Kichineff) read a paper on "Les questions du jour de la psychiatrie sociale." The main questions dealt with were those of organisation of administration on more democratic lines, female nursing of males, and special psychiatric training.

Marriera (Barcelona) read a paper on "L'organisation de la

direction des asiles d'alienés et l'inspection des asiles par l'état." He strongly advocated that the administration of asylums should be under alienists, and gave an account of the arrangements in vogue in Spain.

Other papers that may be mentioned were: Lloyd Tuckey (London), "Some Forms of Mental Obsessions treated by Hypnotic Suggestion"; Muskens (Amsterdam), "Sur la nécessité de diviser des soins pour les épileptiques en deux catégories"; Shinchichi (Tokio), "Eine Beschreibung des Dorfes Iwakoera"; Bezzola (Bodensee), "Du traitement psycho-synthétique des névroses"; Ley (Fort Jaco Uccle), "Le traitement par l'alitement."

Under the impetus of Ferrari (Bologna) and Frank (Zurich) an international association was formed to study the measures that could be adopted for the prevention of nervous and mental diseases.

Special mention should be made of an interesting exhibition, organised by Dr C. E. Daniels, which, though of general medical aims, contained an important section of psychological apparatus and of historically interesting instruments used in the restraint, etc., of the insane.

The social aspects of the Congress were especially well arranged. The Congress was formally opened in the presence of the Queen and Prince Consort, and introductory addresses delivered by Dr Van Raalte, the Minister of Justice, and Professor Jelgersma, the President of the Congress. The Director of the world-famous Rijks Museum conducted parties over the picture galleries. Clinical demonstrations, receptions, concerts, dinners, theatrical representations, all contributed to the general success of the re-union, and there could have been few who did not go away with the pleasantest memories of an instructive and enjoyable gathering ERNEST JONES.

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Review
of
Neurology and Psychiatry

Original Articles

THE MECHANISM OF THE PLANTAR REFLEX, WITH ESPECIAL REFERENCE TO THE PHENOMENON OF CROSSED REFLEX.

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In order to obtain an accepted basis for the discussion of the mechanism of the plantar reflex, certain generally accepted opinions may be briefly enumerated. Under normal conditions, stimulation of the skin of the sole of the foot causes plantar flexion of the hallux, or small toes, or both. We may also observe contraction of the tensor vaginæ femoris (Brissaud's reflex), and a semi-withdrawal of the foot attended by dorsal extension of the whole foot. Flexion of the great toe seems usually to be the first and the most constant response, but occasionally flexion is noted only in the small toes, and such a manifestation seems to be without significance. Capriati regards contraction of the tensor vaginæ femoris as second in point of time, and introductory to the general movement of withdrawal, but this contraction may take the place of the flexion of the toes and yet be without significance. Both the flexion of the toes and the contraction of the tensor vaginæ femoris are reflex, and not under the control of the will. The

general movement of withdrawal of the foot is semi-voluntary, and in most cases can be inhibited by voluntary effort. Under certain conditions the movement of plantar flexion of the great toe is replaced by a slow dorsal extension of the toe, sometimes accompanied by extension and sometimes by flexion of the small toes. The tensor vaginæ femoris contraction under such circumstances remains unaltered. The slow extension of the great toe is the essential feature of this modification of the reflex (Babinski reflex). It is generally agreed that this extension of the great toe on stimulation of the sole of the foot is found only in diseases of the upper portion of the motor tract, that is, the pyramidal tract, or central motor neurones. While it does not occur in so-called functional affections such as hysteria, it is found immediately after coarse lesions that affect the tract, before any definite changes can be supposed to occur in the tract itself. Thus I have seen it within six hours after the injury, in a case of extra-dural hæmorrhage from fracture of the skull, and have noted its disappearance, giving place to the normal reflex, twelve hours later, after the clot had been removed. In such a case the motor tract is put out of function from pressure, yet there are no pronounced structural changes in it of a kind that might be spoken of as organic disease of the tract itself.

The Babinski reflex, however, is not an inevitable accompaniment of organic diseases of the pyramidal tract, for many cases of such diseases with spasticity, exaggerated reflexes, etc., show the normal flexor reflex. The study of the Babinski reflex shows that this normal flexor reflex is dependent upon the integrity of the central motor neurones (5, Fig. 1), and not improbably the special neurones that preside over flexion of the great toe (1, Fig. 1). It seems an inevitable conclusion that the normal plantar reflex is a cerebral and not a spinal reflex, and that the reflex arc is made up of a chain of neurones of which the central motor neurone forms one link. The arc must therefore consist of at least four neurones: a peripheral sensory neurone from the sole of the foot to the bulbar nuclei; one or more central sensory neurones from the bulbar nuclei to the Rolandic region; a central motor neurone from the Rolandic region to the fifth lumbar and first sacral segments; and a peripheral motor neurone from the anterior horns in these

segments to the flexors of the great toe. When conduction from the central motor neurone is impaired by a lesion in any part of its course, the sensory impulse passes directly across the cord, without ascending to the brain, to the cells in the anterior horn, which send fibres to the extensors of the great toe and lie a little higher up than the cells for the flexor muscles. We may assume that the peripheral sensory neurone sends collaterals (6, Fig. 1) to these extensor cells (3, Fig. 1), and that under normal conditions this collateral path is less permeable to the sensory impulse which goes to the flexor cells by way of the cortex. The presence of the Babinski reflex with complete transverse lesion of the cord shows that this collateral path must lie wholly in the cord, and that the Babinski reflex is purely of spinal origin.

Assuming that this hypothesis as to the anatomical composition of the plantar reflex arc be correct, the question naturally arises: why do not breaks in conduction in other neurones lead to a similar diversion of the nervous impulses to the extensor neurones? In lesions of the peripheral sensory neurone, of which tabes affords the simplest type, it is well known that the plantar reflex is unaffected, or it is diminished or lost, according as sensory conduction is more or less affected. Here, with impaired conduction in the sensory neurone, it is not likely that there could be as much conduction in the less permeable collateral going to the extensor cells as in the main fibres of the neurone. When the pyramidal tracts are also involved, as in ataxic paraplegia, there seems to be some possibility of a conduction across to the extensor cells, even with considerable loss of sensibility in the legs, and a Babinski reflex is usually present. Lesions of the peripheral motor neurone are apt to be somewhat extensive, and to involve the extensors of the foot and toes more than the flexors, as in neuritis and in anterior poliomyelitis. This may account for the absence of the Babinski reflex in these affections.

Our knowledge of diseases limited to the central sensory neurones is vague, as is our knowledge of the anatomical position of those neurones. In the cases of anæsthesia from cerebral diseases that I have had the opportunity of studying, there was also more or less motor impairment, so that, when a Babinski reflex was present, it may have been due to the

involvement of the motor neurones. In hysteria the Babinski reflex is not found, but a diminution or complete loss of the normal plantar reflex is not uncommon. Whether hysterical anæsthesia be purely ideational, or whether the sensory projection tract be also involved, is still uncertain. In the former case the reflex arc ought not to be disturbed, but if the projection tract be involved it would indicate that a break here would not produce a Babinski reflex, and, thus far, it would militate against the hypothesis advanced.

A further interesting phenomenon in connection with the plantar reflex has thus far attracted little attention. This is the occurrence of a crossed reflex—a contraction of the muscles on one side from stimulation of the sole of the opposite foot. It is a phenomenon of rather rare occurrence, observable for the most part in patients in whom the semi-voluntary movements of withdrawal are especially lively, and who seem peculiarly ticklish. I have observed in association with it at times an unusual extension of the reflexogenous zone for other cutaneous reflexes; it being possible to obtain the cremaster reflex, for example, by irritating the skin of the lower leg, or even of the sole of the foot.

The presence of a crossed plantar reflex in cases presenting a Babinski reflex is of special interest. The first case of the sort which came under my observation was one of meningeal hæmorrhage with laceration of the brain cortex. There was right hemiplegia with contracture, exaggerated tendon reflex, ankle clonus, and a Babinski reflex on the right side. There was a normal flexor plantar reflex on the left side. Scratching the sole of the left foot caused plantar flexion of the right great toe. The interpretation of this phenomenon is by no means easy. For some time I failed to obtain this crossed reflex in cases of ordinary hemiplegia from capsular hæmorrhage, so I thought that it might be of value in distinguishing between cortical and capsular lesions. It occurred to me that some central motor neurones of the opposite right side of the brain might cross over to the left hemisphere in the corpus callosum, and thus convey the sensory impulse from the left foot to the extensor cells in the right anterior horn of the cord. This hypothesis had to be abandoned later when I found the same crossed flexor reflex in a number of cases of ordinary hemiplegia

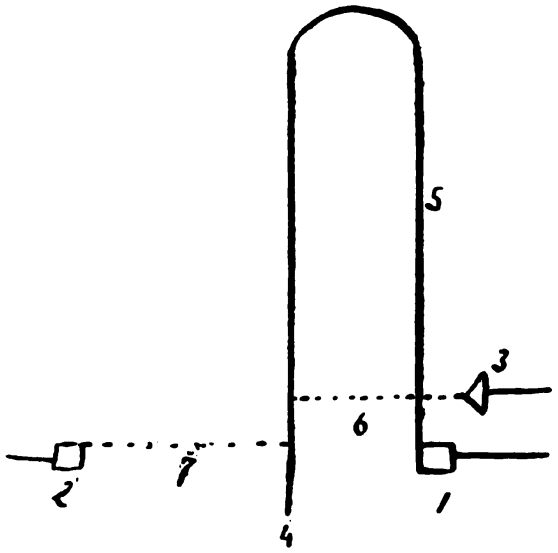

FIG. 1.

1. Motor cells for flexor of great toe.
2. Motor cells for flexor of opposite great toe.
3. Motor cells for extensor of great toe.
4. Sensory neurones.
5. Central motor neurones.
6. Collateral to extensor cells.
7. Collateral to flexor cells on opposite side.

from capsular hæmorrhage, which had a Babinski reflex on stimulating the sole of the paralysed foot. Finally, in a case of ataxic paraplegia with diminished sensibility in the legs, ataxic gait, exaggerated knee-jerks in both legs and a Babinski reflex in each foot, I obtained normal plantar flexion of the great toe in each foot from stimulating the skin of the opposite foot. In a case of this sort the central motor neurones are evidently affected, and apparently, from the existence of a double Babinski reflex, they are no longer capable of playing their part in the normal reflex mechanism. The peripheral sensory neurones are also involved, but apparently there is still some capacity left in them to conduct sensory stimuli. If we assume that in some cases these neurones are hecateromeric, one process passing up on the same side of the cord to the bulbar nuclei, the other crossing over to the opposite anterior horn (7, Fig. 1) to the motor cells for the flexor muscles of the great toe (2, Fig. 1), we have a fairly satisfactory explanation of the mechanism of this crossed flexor reflex, which, unlike the direct flexor reflex, is a spinal reflex.

NOTE.—This paper was presented in substance at the meeting of the American Neurological Association in 1902, before the publication of Dr Bramwell's (1) paper on the crossed plantar reflex. The case cited above, where a flexor reflex was obtained on both sides from stimulation of the opposite foot, yet where there was a Babinski reflex on both sides from direct stimulation, seems a strong argument against the hypothesis of a reflex "set to extension," which Dr Bramwell has advanced as an explanation. Dr Sherrington's diagram (2) of the synoptic connections of neurones as an explanation of the irradiation of a reflex reaction seems particularly applicable to this condition.

1. *Review of Neurology and Psychiatry*, i. 795, December 1903.
2. "The Integrative Action of the Nervous System," p. 155.

FIG. 1.

1. Motor cells for flexor of great toe.
2. Motor cells for flexor of opposite great toe.
3. Motor cells for extension of great toe.
4. Sensory neurones.
5. Central motor neurones.
6. Collateral to extensor cells.
7. Collateral to flexor cells on opposit side.

ARTERIO-SCLEROSIS IN RELATION TO MENTAL DISEASE.[1]

By C. MACFIE CAMPBELL, B.Sc., M.B., Ch.B.

THE presence of arterio-sclerosis in cases of mental disease, especially in advanced life, is comparatively common, and often appears to be of little importance.

In some disorders, however, the mental and physical impairment is intimately related to arterio-sclerotic changes of the cerebral vessels, and it is this group of cases to which attention will be directed. The rôle which arterio-sclerosis plays in the involution, pre-senile, and senile psychoses involves too many general questions to be more than referred to.

Cerebral arterio-sclerosis may be part of a general arterio-sclerosis, or it may exist without other vascular systems being implicated, at least to the same degree. The absence of the signs of general arterio-sclerosis does not, therefore, exclude the possibility of cerebral arterio-sclerosis. The cardinal symptoms of arterio-sclerosis are increased blood-pressure, thickened peripheral arteries, hypertrophy of the left ventricle, with accentuated second sound. When, in addition, the patient complains of headache and dizziness, marked fatiguability and exhaustion, and failure of memory, cerebral arterio-sclerosis is distinctly indicated.

Ophthalmoscopic examination gives still more direct evidence of the condition of the cerebral vessels. In some cases one can even see the indentation of veins by the stiffened arteries, but numerous other changes are important diagnostic aids.

Cases of mental disorder, based on cerebral arterio-sclerosis, fall into two main groups. In the first the symptoms are less severe and more stationary, and Alzheimer calls this the nervous form of cerebral arterio-sclerosis. In the second group are included the more severe and progressive cases, with a varied neurological picture and extreme reduction of mental activity. This second group has been further subdivided, but chiefly upon anatomical grounds, and the clinical differentiation of the various

[1] Read before the American Medico-Psychological Association at Washington D.C., May 8, 1907.

sub-groups is still incomplete. It includes, among other forms, Binswanger's chronic progressive subcortical encephalitis.

The nervous form of cerebral arterio-sclerosis is not uncommon, and many patients are cared for at home or in almshouses. They are frequently described as cases of melancholia, neurasthenia, hypochondria, etc. The onset of symptoms usually occurs between fifty and sixty, but sometimes earlier. The most characteristic symptoms are headache and attacks of dizziness, marked fatiguability and memory defect. The headache is frequent and persistent, and may be influenced by change of blood-pressure, as during straining at stool. Attacks of dizziness are common, especially in relation to change of position. Various subjective feelings are complained of—pressure on the head, flickering before the eyes, buzzing in the ears; alcohol is not tolerated so well. The patient begins to find difficulty with his work, especially in anything that goes beyond the usual routine; his store of memories is not so completely at his disposal as previously; he finds especial difficulty in imprinting new facts on his mind. He is easily fatigued and exhausted, is irritable and depressed, broods over his failing energy and exaggerates his symptoms. The condition shows little tendency to progression, and death is usually due to an apoplectic attack, cardiac disease, or some intercurrent affection, such as broncho-pneumonia. Such cases may show no definite focal symptoms, or they may have transitory attacks of weakness or apoplectiform attacks, leaving a permanent hemiplegia with or without additional symptoms, such as aphasia.

Post-mortem the brain may show little diminution in weight. The striking feature is the marked arterio-sclerosis of the cerebral vessels, both in cortex, white matter, and basal ganglia; focal lesions may be absent. Histological examination shows certain changes in the cortex, but no marked disappearance of nervous tissues.

To illustrate this form of mental disorder based on arterio-sclerosis a brief summary of one case may be given.

The patient was a man of good education, not alcoholic, but with a history of syphilis in early life. About the age of forty-six he became very irritable, felt played out, gave up his work; he was sleepless and restless at night, complained of numerous aches and pains, was depressed, suspicious of his wife,

obstinate. He worried a great deal over his health, thought that he would become insane, dreaded commitment, talked of suicide. He was committed at the age of fifty-five. The cardinal symptoms of general arterio-sclerosis were present, also albuminuria; in addition, he complained of occasional dizziness and faintness, of ringing in his ears and intense headache. The face was smoothed out, the mouth somewhat pouting; he stated that he had occasional difficulty in swallowing liquids, but none was observed. Innervation of the left facial was defective, otherwise there was no circumscribed weakness. His gait was rather shuffling. Speech was careless and slurring, but without the tremor or sticking of the general paralytic. The writing was somewhat tremulous; he wrote "bridiade" for "brigade." On the slightest provocation, or even with none, he would develop a lachrymose facies, but was easily persuaded to smile.

Mentally, he was depressed over his condition, complained of terrible nervousness and weakness, was very easily fatigued by mental operations; his memory was poor, but retention fair.

He described the onset as a gradual breaking down. "I was just like an old man—gradually gave out. I feel like seventy-five; am only fifty-five." He showed keen appreciation of his general impairment, tended to exaggerate physical ailments. After five months in hospital, during which period his condition was stationary, patient had a general convulsion, which was followed by stupor, coma, and death.

Such cases as this have been described symptomatologically as neurasthenic melancholia or hypochondria, but its close relation to cerebral arterio-sclerosis is clear. In addition to the general symptoms—headache, dizziness, attacks of fainting, buzzing in the ears—emphasis must be laid on the slurring articulation, the difficulty in swallowing, the lability of emotional expression. These features at once suggest a similarity to those cases of brain disease where a picture resembling bulbar paralysis is produced by a bilateral interference with the cortical innervation of the bulbar nuclei for movements of the lips, tongue, and pharynx, *i.e.* cases of pseudo-bulbar paralysis. One of the most frequent causes of this syndrome is arterio-sclerosis of the cerebral vessels. It is impossible to do more than touch the topic of explosive laughter and crying in such patients as the one just reported. Typical unmotived explosive laughter may

occur; facile tearfulness, with little affect, is also found. In some cases, however, the affect itself is somewhat explosive, and not merely the facial expression, and the amount of discrepancy between real affect and emotional expression is variable.

To discuss this question adequately would involve an examination of the mood in the various depressions in advanced life, and this cannot be done here.

The points already referred to show that the group of cases described above has relations on the one hand with the various depressions in advanced life; and, on the other, with organic brain disease of vascular origin. In emphasizing the arterio-sclerotic element in the picture, one ought not to fail to do justice to the other element—that element which has received prominence in the older symptomatological grouping of the cases as melancholia and hypochondria. This must be kept in mind in view of various unsolved problems. Why is cerebral arterio-sclerosis in some cases compatible with good mental and physical health, while in other arterio-sclerotics strength and initiative and the power of assimilation undergo marked impairment and the patient develops an abnormal mood and morbid ideas? In these latter cases what determines the onset of the impairment? The arterio-sclerosis is not acute, but the symptoms may develop rapidly. The exact part played by circulatory variations and renal insufficiency in this disorder is well worth further investigation. The literature does not contain many cases which have been fully studied from this point of view.

In the nervous form of cerebral arterio-sclerosis the mental level of the patient may remain practically stationary for years, although slow progressive decline, varied by little episodes which may necessitate commitment, is common. In some cases of cerebral arterio-sclerosis the patient may go down-hill with surprising rapidity, presenting a picture of extreme general feebleness and confusion. Such was the course in the case of a man of fifty-one, with slight cardiac hypertrophy and mitral incompetence, with no albuminuria, whose brain presented a very definite arterio-sclerosis. No adequate cause could be found for the extremely rapid decline in this case.

The cases of still more pronounced mental impairment, based on arterio-sclerosis, are grouped by Alzheimer under the title, Severe Progressive Arterio-sclerotic Brain Degeneration. The

disorder may here begin with the same symptoms as in the nervous form, but instead of remaining at the same level the patient, if he does not succumb to one of a series of attacks, may be reduced step by step to a state of the most profound dementia, in which vegetative condition he may exist for a considerable time with occasional unexpected flickers of intelligence.

In the early stage, as in the nervous form, there is restlessness, irritability, a depressed and tearful mood, considerable mental sluggishness, difficulty of recalling and using the store of memories, impaired ability to receive and retain new impressions. As the disease progresses the mental processes become still more sluggish, the patient's store of ideas becomes more and more impoverished, elaboration of new material ceases, life becomes almost vegetative. From the first one notices that the mental possibilities of the patient show considerable variation from time to time; and even in the advanced stages there are brighter moments, when he gives evidence of transitory grasp of environment or may utter fragmentary references to past incidents. The persistence, even in the latest stages, of insight into his unfortunate condition is very striking.

The dementia reaches a degree comparable to that of the general paralytic, but it is rather an extreme reduction than a total disintegration; the elements left are fragmentary, but not distorted nor grotesque.

Such is the general scheme of the mental decline, but this is diversified by various morbid ideas, usually of a depressive or persecutory nature; from time to time there are delirious incidents, episodes of bewilderment or uneasiness, attacks of excitement with delusions and hallucinations. The history of the physical decline may be very complicated and include attacks of different kinds, with a great variety of residual defect symptoms. In these cases one has the opportunity of studying hemiplegic attacks, transitory or permanent hemianopias with the phenomena of conjugate deviation, aphasias, asymbolias, and a great variety of irritative phenomena. The neurological episodes include attacks of dizziness, general epileptiform attacks, apoplectiform attacks, attacks confined to localised twitching. During these latter attacks the patient may be quite conscious and converse with the physician, or he may only make frag-

mentary delirious utterances, or consciousness may be lost altogether.

The resulting defect symptoms give rise to a very complicated neurological status. In addition the pupils may be sluggish or quite rigid. The speech is usually slurring. A moderate lymphocytosis of the cerebro-spinal fluid is sometimes found.

The brain shows a more severe degeneration than in the nervous form of cerebral arterio-sclerosis. The weight is usually diminished, and the white substance shows very pronounced changes, while numerous focal softenings may be present.

Clinically it may be very difficult to diagnose such a picture from dementia paralytica with focal symptoms, and from senile dementia with focal symptoms. In general, as Alzheimer says, the paralytic and senile dement strike one as insane; while with the arterio-sclerotic dement the "crazy element" in the picture is less striking. The physical signs of general paralysis with the crazy ideas and general mental disintegration usually enable the diagnosis to be made; while the typical senile dement, with focal symptoms, shows a distinctive mental picture, in which disorientation, a tendency to confabulation and crazy ideas are prominent features.

These general diagnostic points are, however, insufficient, and the relation of the senile psychoses to arterio-sclerotic dementia is complicated. Even patients presenting the less severe form of cerebral arterio-sclerosis may have episodes which are indistinguishable from those of the ordinary senile dement. We know little about the ætiology of senile dementia and of its various forms. It is possible that in certain cases of arterio-sclerosis there is a combination of phenomena due to the vascular changes with others of so-called senile origin.

The term arterio-sclerotic dementia is justified by the prominence both clinically and anatomically of certain changes of vascular origin, but it must be emphasised that the whole picture is very complex, and that the complication of arterio-sclerosis with senile or other changes is not to be excluded.

Correlation of definite focal lesions with certain neurological symptoms is possible, but any detailed correlation of mental defects with anatomical changes is premature.

A patient at present under observation illustrates well the

limits of our knowledge with regard to the organic dementias. He is a man of sixty, profoundly demented, who now only shows sign of mental activity by uttering occasionally an almost inarticulate delirious remark.

At the age of forty-five he began to fail mentally; at the age of fifty-eight he had a general convulsion, and was admitted to hospital. During the two years of hospital residence he has shown progressive decline, such as one meets in severe progressive cerebral arterio-sclerosis; he has manifested no delusions, and has even in the late stages shown remarkable persistence of insight into his general impairment. The neurological history has been varied; attacks of different kinds have been frequent. Right-sided hemianopia and right-sided sign of Babinski are permanent residuals from attacks in hospital. Numerous attacks consisted in transitory left-sided hemianopia, irritative and paralytic phenomena in the left face and arm, and anæsthesia of the same region. The knee-jerks have been absent since admission to the hospital; the cerebro-spinal fluid shows a well-marked lymphocytosis; the sign of Romberg has been observed. Since admission the pupils have become more and more sluggish and somewhat irregular; the speech is extremely slurring, but not suggestive of general paralysis.

Peripheral arterio-sclerosis is well marked, with high blood-pressure and cardiac hypertrophy; there is no evidence of renal insufficiency.

The general nature of the mental reduction is similar to that in severe progressive cerebral arterio-sclerosis. The irritative and paralytic phenomena are compatible with cerebral arterio-sclerosis, but our present knowledge of such attacks has too little definition for them to give much aid in the diagnosis.

The absence of knee-jerks, sign of Romberg, marked lymphocytosis of the cerebro-spinal fluid, sluggish, irregular pupils, force one to reserve the diagnosis; for it is not as yet sufficiently established what weight is to be laid on these signs, individually and collectively, in the clinical differentiation of the organic dementias.[1]

The presence of epileptiform convulsions in cerebral arterio-

[1] The patient died on May 7, 1907. Post-mortem examination showed very pronounced cerebral arterio-sclerosis, the left visual cortex being destroyed by a large area of softening. On microscopical examination the typical histopathological changes of general paralysis were found.

sclerosis has already been mentioned. In some cases these convulsions are the most prominent feature of the disease. The patient may show no mental impairment, or the convulsions may occur along with other characteristic physical and mental symptoms. That the circulatory system has an important relation to the convulsions is shown in some cases by the results of treatment of the cardio-vascular disorder. The fact that in certain arterio-sclerotic epileptics compression of the carotids may bring on convulsions points in the same direction.

In young epileptics with valvular lesions the occurrence of fits in positions which embarrass the circulation, and the reduction of the number of fits under cardiac stimulants, confirms this point of view.

That renal incompetency may be in some cases an important element is possible, but well-marked cases of senile epilepsy may show no evidence of kidney trouble. Even though the cardio-vascular disorder may explain the occasion for the occurrence of the fit, it does not explain why only certain individuals should give this reaction to the disease. This question is quite obscure, and one must admit that the ætiology of senile epilepsy is in the same condition as that of epilepsy in young life. As to the general symptomatology of those cases of senile epilepsy with pronounced arterio-sclerosis, in the intervals between the fits the patient may show little defect, or he may show, in more or less pronounced degree, the symptoms characteristic of the nervous form of arterio-sclerosis. The attacks may not be limited to general convulsions.

A patient sixty-three years of age, at present under observation, has, in addition to typical epileptic fits, shown a variety of attacks without any motor implication. The attacks have been of the nature of a transitory loss of consciousness of a few moments duration, or of transitory confusion, or of a "Dämmerzustand" or delirious episodes, or of episodes of exaggerated irritability, suspicion, and indignation with ideas of reference, and possibly hallucinations.

This patient has excellent insight between the attacks, and is keenly aware of his poor memory and retention; he has a permanent tendency towards ideas of reference. Of late he has developed certain ideas of greatness a rather unusual feature.

SUMMARY.

I. While arterio-sclerosis of some degree is a commonplace finding in cases of mental disorders, especially in advanced life, there are cases where the cardio-vascular disorder seems to be the central element in the clinical and anatomical picture.

II. Certain cases symptomatologically described as melancholia, hypochondria, neurasthenia, etc., may be better grouped on etiological grounds as cases of arterio-sclerotic brain disorder; while the arterio-sclerosis is an important factor, the factors which led to the symptomatological grouping are not to be neglected. This necessitates the analysis of the depressions in advanced life.

III. In certain cases of organic dementia the arterio-sclerotic changes are obviously the most important element in the process, and the term arterio-sclerotic dementia is justifiable; but the relation of certain mental symptoms to similar ones in the pre-senile and senile psychoses must be kept in mind. This necessitates the analysis of the pre-senile and senile psychoses.

It is premature to correlate the whole symptomatology with the arterio-sclerotic part of the anatomical findings.

IV. The neurological picture in advanced cerebral arterio-sclerosis is still ill-defined; and for the differential diagnosis of the various organic dementias further clinical material is required.

V. In certain cases of epilepsy with onset in late life, the convulsions and general symptomatology are closely related to arterio-sclerosis.

Abstracts

ANATOMY

THE NEURONE DOCTRINE. (La doctrine du Neurone.) CAMILLO (429) GOLGI, *Nord. Med. Arkiv.*, Afd. ii., H. 1, 1907.

THIS paper contains a powerful attack on the doctrine of the neurone, of which Golgi remarks that on all sides it is now affirmed to be tottering to its fall. He states that the present conception of this doctrine is as follows:—

1. The neurone is an embryological unity, derived from a single cell.

2. It is anatomically, even in the adult state, a cellular unity.

3. It is a physiological unity.

These three points constitute the fundamental conception of Waldeyer; they have, however, been enlarged by accessory propositions on the anatomical and functional side, *e.g.*—

4. The relation between neurones is by contact only.

5. With the exception of neurones there exist scarcely any nervous elements.

6. The neurones are trophic units.

From the physiological point of view the neurone doctrine has found its most perfect expression in the theory of dynamic polarisation, according to which nervous currents pass cellulipetally in dendrites, and cellulifugally in axons. Cajal has from time to time modified this theory, so that it may harmonise with the march of the nerve currents in the spinal ganglia cells, where, according to him, the currents pass directly across the T piece, avoiding the nerve cell. Golgi admits that the theory of dynamic polarisation is not an essential part of the neurone doctrine, although it is very intimately bound up with it. He does not agree with Cajal that the peripheral branch of the spinal ganglia cell is to be identified with a dendrite, and that the possession of a myeline sheath is only a secondary fact, rendered necessary on account of the length of this branch, and he points out that it is easy to demonstrate at the T junction the passage of both peripheral and central fibrils into the cell body.

In the same manner he cannot admit as a decisive argument the affirmation, one of the main points in support of the theory, according to which the branches of the small cortical cells in the molecular layer of the cerebellum terminate on the bodies of the Purkinjé cells, for he has shown that the neurofibrils coming from the axons of these small cells are continued on into the rich and very characteristic network existing in the granular layer, and to which he has given the name of the diffuse nervous network, attaching to it the greatest importance. Its formation is contributed to by all the nervous elements of the central nervous system.

Moreover, he does not see how dendrites can be exclusively concerned in the reception of nervous excitations, when they can be traced not only to the surface of convolutions, but even beyond the molecular layer and into the depths of the meninges. He proceeds to discuss the three fundamental conceptions of the doctrine.

1. *The neurone as an embryological unity.*—The basis of this conception comes from the studies of His on the development of the

nervous system. But His' results have been controverted by other observers, *e.g.* the work of Capobianco and Fragnito on the pluricellular origin of ganglion cells, and those by Balfour, Beard, Bethe, etc., on the derivation of nerve fibres from chains of cells, all of which are opposed to the neurone doctrine. Golgi himself, whilst he does not consider that these results afford valid arguments against the doctrine, on the other hand does not attribute a demonstrative value in favour of the doctrine to the recent researches on the central origin of regenerated fibres. They do not, in his opinion, justify an affirmation more ample than the central origin of nerve fibres—a phrase far from having the signification of the origin of each fibre from a corresponding cell. In the present state of our knowledge of the histogenesis of the nervous system it is not, he thinks, possible to affirm with certainty that what we know of the origin of nerve cells has any fundamental value serving to support the supposed independence of the nerve cell.

2. *The neurone is even in the adult state an independent cell unit.*—In this section Golgi gives an account of his conception of the structure of the nervous system. It is unnecessary to recapitulate his account of the two classes of nerve cells, so well known as Golgi's cell, type 1 and 2. The great interest of this section centres around his description of the diffuse nervous network which is found in all layers of the central grey matter. He considers that it is an anatomical entity, and not merely a hypothesis, and that it plays a fundamental rôle in the specific functions of the central nervous system. He holds that there is no necessity to insist on direct continuity between the fibrils of different regions as a condition *sine qua non* for the transmission of the excitation from one to the others; but that the formation of the network, and, above all, the fact that *all* the elements of the central nervous system share in its construction, proves the anatomical and functional continuity between nerve cells, and for this reason he cannot admit the idea of the independence of each nerve cell, which is the essential basis of the neurone doctrine.

There are many facts which, in his opinion, support the idea that the dendrites serve *partly* as organs of nutrition for the cell body—one of these has already been given, another is derived from the behaviour of the blood-vessels in the cells devoid of dendrites—and he cites the existence of blood-vessels inside the body of the giant cells of Lophius piscatorius, and the enclosure of the Deiter's cells beneath the Corpora bigemina by a band of capillaries. He mentions also certain anatomo-pathological and experimental facts which favour the idea. He has always contended, since they are direct emanations from, and of similar structure to, the cell body, that they are able to participate in the specific function which is attributed to the nerve cell.

Apáthy's results demonstrating the passage of neurofibrils from one cell to another, as well as the existence of a diffuse elementary network, imply the demolition of the neurone doctrine, and give express confirmation to the existence of a diffuse nervous network, and to the doctrine which has been raised on this fact, but Golgi utters a warning against transferring deductions derived from invertebrate structures to vertebrates.

He refers to the studies of Bethe and Donaggio on neurofibrils, but does not believe we may identify the fibrillar or reticular structures of these two authors with the structures shown by Apáthy in the nerve cells of worms; nor will he admit that all the fibrils and reticular structures, especially the internal structures described by them, represent respectively neurofibrils and networks of nervous nature.

3. *The doctrine of the physiological independence of the neurone,* it is affirmed, is included in the law of Waller, and, from the results of experiments on systematic degeneration of nerve centres, in the doctrine of dynamic polarisation. Golgi states that the argument which has been formed on Waller's law is, in his opinion, absolutely devoid of value; it cannot be used with any validity against the idea of the existence of intimate relationship between nerve cells by means of the diffused nervous network. As for the doctrine of dynamic polarisation, it is evident that it is but one of the expressions of the physiological conception, and does not exclude the possibility of others.

The doctrine of the fundamental independence of the neurone may find indirect support from studies on cerebral localisation, provided that the idea of localisation retains its original form, attributing to well-marked and limited regions of the brain sensory and motor functions quite distinct and precise. But the idea of localisation has to-day undergone profound modification. Experience shows with what little precision one is able to limit central excitable zones, and the discovery of collaterals raises fresh difficulties. All the facts indicate that nerve fibres may have relation with an infinite number of nerve cells, even in parts of the nerve centres entirely different and widely removed from one another. He alludes to his previous works on the subject, illustrated by drawings which show the default of all the anatomical conditions necessary to give a foundation to the physiological conception of localisation.

Whilst declaring as inadmissible the existence of sharply defined central districts, representing the exclusive seat of the central distribution of nerve fibres, he believes, however, that we must admit that territories exist where certain fibres are most directly distributed in preponderating numbers.

He has repeatedly contested that the specific function of the

nervous system is not in relation with particular organisations of nerve centres, but rather with specificity of peripheral organs destined to receive and transmit impressions.

In Golgi's opinion not one of the arguments on which Waldeyer has supported the doctrine of the individuality and independence of the neurone will bear examination.

He has never had occasion to abandon the idea on which he has always insisted, that the nerve cells act in harmony, so that one is obliged to suppose that several groups of elements exercise an accumulative action on the peripheral organs through the intermediation of integral bundles of fibres. JOHN TURNER.

STRUCTURE AND CONNECTIONS OF THE NEURONES. (Structure et connexions des neurones.) (430) S. RAMON Y CAJAL, *Nord. Med. Arkiv.*, Afd. ii., H. 1, 1907.

THIS important and beautifully illustrated paper bristles with discoveries, new within the last two years, connected with the structure of spinal and sympathetic ganglia cells, and of regenerating nerves, made by means of the author's reduced silver method.

In the author's opinion they afford strong additional evidence in favour of the neurone doctrine.

The first twenty pages contain a résumé of Cajal's monumental work on the structure of the nervous system. As the result of these researches he arrives at a conception which comprehends the following propositions:—

Nerve cells are morphological individualities, neurones.

The nerve elements possess reciprocal relations of contiguity, and not of continuity. The relations of contact, more or less intimate, always establish themselves, not between the nervous arborisations alone, but between these ramifications on the one hand, and the body and dendrites on the other hand. These results imply three physiological postulates:—

1. That currents are transmitted from one element to another by means of a kind of induction or influence at a distance.

2. That the cell bodies and dendrites, as well as axons, are apparatuses of conduction, since they represent links between the afferent nerve fibres and the axons.

3. An examination of the march of impulses in the sensory organs proves not only that the dendrites play a conducting rôle, but further that the nervous current is in the dendrites, cellulipetal or axipetal, whilst in the axons it is cellulifugal (law of dynamic polarisation).

Proofs of the neurogenetic doctrine of Kupffer and His.—Cajal's results with reduced silver show that at no time in the evolution of the axon does one come across cellular chains (catenary theory) nor discontinuous axons; on the contrary, with both embryos and nerves in process of regeneration the new fibres produce themselves at the moment of budding of the axons, and in perfect continuity with motor or sensory neurones. The supposed peripheral neuroblasts disposed in chains represent late formations always distinct from the protoplasm of the axis-cylinders, and probably belong to the mesoderm.

Proofs collected from the mechanism of regeneration of nerves.—He describes among the appearances seen in the central end of the cut sciatic nerve of an animal (rabbit, cat, dog several weeks old), killed three days after the section, the terminal buttons on the divided axons (Perroncito met with them within two days after the lesion), sometimes very large and irregular, and apparently at first naked, but in the course of four to six days enveloped in a nucleated connective tissue capsule. Also a very curious phenomenon, to which he gives the name of the "apparatus of Perroncito," consisting of the unravelling and dispersion of the neurofibrils of the axons, followed by the early appearance of a bundle of young nerve fibres, of which some, enlarging and ramifying very actively, give place to a system of spiral filaments which enclose the rectilinear and axial bundles of neurofibrils without leaving the sheath of Schwann. He regards this mechanism as a pathological new formation, but it is easy to see how it, as well as the precocious budding of nerve fibres from the central end, and above all the presence of bulbous enlargements on the end of axons penetrating into the cicatrix, are facts of great value in favour of the neurogenetic doctrine of His, because all these appearances are anterior to the phase of multiplication of the cells of Schwann.

He has repeated the experiments of Bethe, etc., of inserting an obstacle between the cut ends of the nerve to prevent immediate union, and finds *often* (not always), after three or four months, a very advanced regeneration in the peripheral segment, and that the intercalated tissue, supposed by the opponents of the neurone doctrine to be devoid of myelinated fibres, contains them in considerable numbers, in the form of a very loose plexus, binding together, across all the obstacles, the central and peripheral ends. In the peripheral segment the nerve fibres divide and produce the very complicated nerve clusters described long ago. Of these secondary branches some run into the bands of Büngner, others in the interstices between these latter, and terminate at different levels by means of a button of growth. The existence of all these ramifications from fibres coming from the cicatrix, and above all

the presence at the end of each branch of a button of growth constantly orientated towards the periphery, are phenomena absolutely irreconcilable with the catenary hypothesis. Furthermore the most delicate fibres found in the bands of Büngner never appear discontinuous, but in continuation evidently with those coming from the cicatrix, and are therefore independent of the cells of Schwann and of the fatty masses of old nerve tubes.

Phenomena of balls.—Besides regenerative processes there are evidences of spontaneous nervous new formation in the nerve centres of adult man. Cajal has shown recently that the body as well as the principal prolongation of a certain number of sensory and sympathetic corpuscles constantly emit nerve fibres which terminate at variable distances, some under the capsule, some in the thickness of the white matter of the ganglion, by means of a button of growth surrounded by a nuclear envelope.

Before undertaking his researches on the regeneration of nerves he thought that these curious appendages represented permanent structures belonging to this category of cells; but now that he has seen them in the great sympathetic of the aged, in the cerebellum, and the ganglia of animals attacked by rabies, and other infectious maladies, etc., he does not doubt but that they represent the result of a process of nerve production, in certain limits a physiological phenomenon, but exaggerated after toxic and other conditions. He does not stop to speculate on the interpretations which this discovery suggests, but limits himself to the statement that the corpuscles with "boutons" or balls afford a brilliant demonstration of the autonomous regeneration of adult neurones, and of their capacity to produce new fibres by simple budding, and without the aid of the cells of Schwann.

Proofs collected from embryonal neurogenesis.—He cites the following facts in confirmation of the neurogenetic conception of His, Kupffer, and Kölliker, based on appearances shown by reduced silver nitrate in the nerve fibres of the embryo chick, after sixty hours of incubation.

1. The neuroblasts of His show in their classical form. The neurofibrils, the only parts of the cell which attract the silver deposit, begin in the cone of origin of the axon, and extend as bundles to the cone of growth, in the interior of which they terminate as a brush. No traces of cell chains, or of bizarre colonies of anastomosing neuroblasts were seen.

2. The motor and sensory nerves (at the second day of incubation) are not composed, as maintained by Beard and others, of series of neuroblasts, but by the continuous and very thin prolongations issuing from the neurones, which march, some independently, others united into bundles, across the intercellular mesodermic spaces. The marginal cells (lemmoblasts of Lenhossék)

appear later (fourth day), and are situated around the nerve bundles, to which they have no relation except contiguity.

3. One can see in the depths of the mesodermic tissue that each fibre of the end branches of the motor and sensory roots terminates free by means of a button of growth exactly like those observed in the nerve tubes in process of regeneration. In these very thin and therefore very recent peripheral branches one never sees any trace of cell chains, nor even of marginal or terminal corpuscles.

4. The motor nerve cords, examined in the neighbourhood of the facial and hypoglossal centres, etc., are entirely devoid of contained nuclei.

5. Also, as is well known, all the central paths are formed without the aid of the cells of Schwann, and by means of a process of growth continued from the axons of associational neurones. To this fact he also adds that not only by the aid of reduced silver nitrate, but also by ordinary methods of coloration, in certain tracks, as those of the cerebellum, a great number of young nerve fibres in process of growth, and provided with terminal buttons, are often found, even in very developed fœtuses (dog, cat, and rabbit).

The neurogenetic theory of His is in his opinion the inevitable postulate that springs from all these observations, and from Cajal's remarks it would appear as though he regards this theory (and by implication the neurone doctrine) as a sacred institution, and any attempt to call in question the truth of it, a presumptuous, if not indeed blasphemous, act.

JOHN TURNER.

(431) **NOTES ON THE WEIGHT AND THE CONVOLUTIONAL PATTERN IN SEVEN CHINESE BRAINS.** P. HARPER, *Archives of Neurology*, 1907, p. 201.

THE paper consists of the summaries of the results of the examination of the brains, and deals with—

1. The weight of the various brains. Only in two cases does the weight come up to the European average, but relatively to the cerebellum the cerebrum exceeds the average owing to the large size of the frontal lobe.

2. The peculiarities of the fissure of Sylvius and the effect of the large frontal development upon the Sylvian angle.

3. The arrangement of the convolutions and sulci in the frontal lobe, the large size of which is chiefly due to the great development of the middle and inferior frontal lobules.

4. The fissure of Rolando—which at its upper end is set far back on the supero-mesial margin. The increase of size of the

frontal lobe which would follow from this position is somewhat negatived by the small Rolandic angle, which only averages 59.4°.

5. The parietal lobe, which in all but one brain is relatively small.

6. The differences in length and mode of termination of the calcarine fissure.

7. The limits of the line of Gennari and the formation of the external parieto-occipital fissure.

8. The sulcus lunatus, which is very clearly represented in seven of the hemispheres. E. B. JAMIESON.

(432) **EXPERIMENTAL INVESTIGATIONS ON THE ARTIFICIAL MALFORMATIONS AND HETEROTOPIAS IN THE SPINAL CORD. (Ricerche sperimentali sulle malformazioni ed eterotopie artificiali del midollo spinale.)** GARBINI and REBIZZI, Perugia. *Premiata Tipografia*, Umbra, 1907.

THE question dealt with in this paper is the possibility of artificial lesions of the cord simulating alterations in development. The authors after experiment affirm that artificial heterotopias and malformations cannot be caused by ordinary manipulation, but are induced by severe injury, *e.g.* severe compression, bending at an acute angle, dropping a weight of 50 to 100 gr. from a minimal height of 15 cm. upon the cord, etc. Artificial malformations cannot be confused, however, with developmental ones on account of their special characters, which are as follows: (1) In artefacts there is a sudden passage from the normal to the abnormal in a single section of the cord, the fissures are displaced, and the segments of cord immediately above and below the heterotopia—peripheral or extra-medullary—appear of less volume than normal. The white and grey substances are mixed in a disorderly fashion. (2) The pia-mater is always ruptured on the side opposite to the point of impact, and here the nerve substance escapes. (3) Very frequently the grey matter is more or less deformed, *e.g.* simulating on one side hemiatrophy; but here the grey matter on the opposite side would appear crushed, and in the sections near the deformity the normal volume of white and grey matter increases in proportion to the extent of the artificial hemiatrophy. (4) The intramedullary part of the root fibres is ruptured, angular, recurrent, and intersecting. (5) Fibres and bundles of fibres are deviated in all directions, tend to intersect, form whorls and figures of 8. Artificial heterotopias at the periphery (or extra-medullary) are cup- or fan-shaped, and most usually are not covered by pia. The fibres are vorticose or tortuous, and may have portions of grey matter scattered amongst them. (6) By Weigert's method one sees

amongst the fibres of the grey matter very fine droplets due to fine fragmentation of the myelin. (7) By Marchi's method fine droplets are scattered throughout the section, and large masses simulating early degeneration at the periphery and anterior columns. The fibres of an extra-medullary heterotopia always appear as if degenerated. Wherever the injury has been most severe, it is there the large irregular drops are most prevalent. Where the cord is lacerated, the margins of the fissure are outlined by large irregular black droplets. (8) Entire groups of cells may disappear. Injured cells are tortuous and broken, bent at sharp angles, or the processes separated from the cell body. (9) The tissue shows spaces, and fissures with torn edges in which the axis-cylinders are bare. (10) The central canal may be dilated, ruptured, apparently double and communicating with artificial cavities and fissures in other parts of the cord. Torn medullated fibres may project into its lumen. DAVID ORR.

PHYSIOLOGY.

OBSERVATIONS UPON THE VOLUME OF THE HUMAN BRAIN
(433) **UNDER VARYING CONDITIONS. (Neue Beobachtungen über Volumschwankungen des menschlichen Gehirns bei bestimmten Einwirkungen.)** E. WEBER, *Monatsschr. f. Psychiat. u. Neurol.*, Sept. 1907, p. 218.

MENTAL work is stated by all who have investigated the subject to cause increase in the volume of the brain. Variations, more or less regularly rhythmical, have also been observed during the continuance of the work. It has been ascertained by the author that under certain circumstances mental work causes only diminution of the brain volume without preceding increase.

The observations were made upon a boy of 10, who had, in consequence of an accident, a cranial defect on the left side of the forehead which afforded an opportunity of recording the brain volume and pulse in the cerebral vessels. The experiments were made as follows. The boy read attentively for a certain time in a book of nursery tales, or counted dots scattered irregularly over a sheet of paper. When the boy began the work in an unfatigued condition the brain volume increased in the usual manner until the conclusion of the work, when it diminished. The pulse also was augmented during the experiment, indicating active dilatation of the pial vessels. After the brain volume had sunk on cessation of work, a second less lasting increase frequently occurred. When the work was continued for a longer period, or if the boy was fatigued at its commencement, as for example after a meal, only

slight volume increase was observed, which disappeared in a short time, the brain becoming less in size than at the beginning of the work. The accompanying marked diminution in amplitude of the pulse pointed to active constriction of the pial vessels. This persisted for a considerable time after conclusion of the work. When the experiments were long continued, no initial increase of volume appeared. An explanation of the difference between previous results and those now recorded may be found in the fact that former observers have employed adults as subjects of experiment. It is obvious that the child's brain is easily fatigued by mental effort, and the accompanying yawning, etc., make it evident that fatigue is associated with the observed diminution of brain volume.

The author correlates his results with Verworn's theory of Biotonus, and considers that the lessening of the cerebral blood supply caused by vaso-constriction prevents undue and harmful dissimilation of the cortical cells in the youthful brain. Vaso-constriction is more marked in the afternoon that in the morning.

In another communication the author has shown that stimulation of certain parts of the nervous system causes first increase, then diminution of the brain volume in animals. Probably vaso-constriction and dilatation of the vessels under normal conditions are dependent upon nervous impulses rather than upon the direct influence of metabolism products on the vessel walls.

Further experiments were carried out with the object of determining the relation between the blood supply to the brain and to the external parts of the head when definite voluntary movements were performed. The ears were enclosed in plethysmographic capsules, the brain volume being at the same time recorded, and the boy was instructed to press the hand of the observer forcibly. Curves were thus obtained which demonstrate that, while the brain volume increases during such movements, the volume of the ears lessens. The author has also shown that such decrease of the blood supply to the ears accompanies the conception without actual performance of voluntary movements in hypnotised subjects, and it is to be presumed that here also there is increase of brain volume. Similar results are obtained by stimulation of the motor areas in animals. W. A. JOLLY.

THE FUNCTIONS OF THE TRIGEMINAL NERVE. H. MORRISTON (434) DAVIES, *Brain*, Summer 1907, p. 219.

IN this paper the distribution and nature of the fibres composing the trunk of the fifth cranial nerve are considered in detail in the light of previous work and of the material obtained by the personal investigation of thirty patients from whom the Gasserian ganglion

had been removed for trigeminal neuralgia. Most of the patients were examined on several occasions and at intervals after the operation varying from a few days to seven years. In this way it was possible to investigate with precision the changes which occur in the field of anæsthesia left by the section of the nerve.

The chief conclusions may be summarised as follows. In a certain number of cases a considerable shrinkage of the area of anæsthesia occurs. Such change is always at an end in six months after the operation, and usually in two.

The cutaneous distribution of the nerve shows considerable variations in different individuals. All these varieties can be classified into four well-marked characteristic types differing very definitely from one another.

The distribution to the external auditory meatus and to the mucous membrane of the mouth also varies within rather wide limits, but is not classifiable into types so definitely and so precisely marked off.

There is no perceptible correlation of the extent or form of the area of supply in the face, the auditory meatus, and the mouth.

The whole of the tympanic membrane is supplied by the trigeminal in about a quarter of the cases, part of it in another quarter, and none of it in about half.

The evidence as to the conveyance of gustatory fibres by the trunk of the trigeminal when examined in detail leads to the conclusion that such a course is certainly not the normal one.

The motor distribution is entirely confined to the muscles of mastication. Experimental work on animals, the effects of the section of the nerve in man, and the results of exploration of the cavum Meckelii with the faradic current to ensure that the removal of the ganglion has been complete, all combine to show that the fifth nerve does not supply the tensor palati muscle.

The association of trophic affections of the cornea with lesions of the fifth nerve is left for discussion in another paper.

AUTHOR'S ABSTRACT.

PATHOLOGY.

THE NEUROFIBRILLARY RETICULUM OF THE MOTOR CELLS
(435) **OF THE SPINAL CORD IN EXPERIMENTAL TETANIC INTOXICATION. (Ancora sul reticulo neurofibrillare delle cellule motrici del midollo spinale nella intossicazione tetanica sperimentale.)** TIBERTI, *Riv. di Patol. nerv. e ment.*, Agosto 1907.

THIS is a sequel to a paper published by the author in 1905.

Numerous observations have shown that the neurofibrillary

reticulum of nerve cells possesses a considerable power of resistance against toxic agents, much greater than that of the chromophile elements.

In fact the reticulum is sometimes found intact in morbid conditions when the chromophile elements have almost entirely disappeared.

The researches of Donaggio and others have shown that the reticulum shows signs of morbid change much more markedly if subjected to the action of two noxious agents such as toxin and cold, starvation and cold, or toxin and starvation, than when exposed to one of these alone.

In his examination of the motor cells of the spinal cord in experimental tetanic intoxication, the author has not found, by Donaggio's method or that of Ramon y Cajal, that the poison exerts any particular action on the endocellular reticular formation. Nothing is seen which can in any way explain the pathogenesis of the tetanic contractions. R. G. Rows.

NEURONOPHAGY IN TRANSPLANTED SPINAL GANGLIA.

(436) **(Neurophagie dans les greffes de ganglions rachidiens.)** NAGEOTTE, *Revue Neurolog.*, No. 17, 1907, p. 933.

THIS paper is a continuation of the author's work published in previous numbers of this journal, and opens with a reference to his own and Marinesco's articles on the subject.

Twenty-four hours after transplantation the cells at the periphery of the ganglion alone survive; those in the centre die, but not from want of oxygen. The cells which survive the first few hours of interference attempt regeneration, then atrophy, and disappear later; but in this paper only the phagocytic phenomena following early death are dealt with, *i.e.* within the first few hours of transplantation.

The polynuclear cells are specially abundant in the centre of the graft, and possess no particular interest at present. Of the *satellite* cells there are two varieties, each differing in morphology and in behaviour. The *endothelial* cells lining the nerve cell capsule proliferate to form a mass occupying the former site of the dead cell; these residual masses disappear in time. *Cajal's cells*, which are purely phagocytic, are situated under the endothelial layer, and with their processes surround the nerve cell. They invade and tunnel it in all directions, and when intracellular digestion is complete, it is no longer possible to distinguish one variety of satellite cell from the other. Both show marked fatty changes; the polymorphs never. The fatty granules are round and numerous, stain badly with osmic acid, are brown in Flemming's solution, and soluble in xylol.

The exact topography and orientation of the excavations made by the phagocytes in the nerve cell are best studied in sections coloured by Cajal's photographic method. They are cylindrical and form a network in the cell. The cell periphery is comparatively free from this network, and only shows a few canals arranged perpendicular to the surface. Such appearances are reminiscent of the canaliculi found in the normal cell, and of the internal network of Golgi. Perhaps it is by this canal system that Cajal's cells enter the nerve cells? Or the network contains some substance for which Cajal's cells have a special affinity? Or it may be formed of the protoplasmic processes of Cajal's cells, and in pathological conditions the cell body is simply guided along the prolongation? DAVID ORR.

CLINICAL NEUROLOGY.

SYRINGOMYELIC BULBAR DISEASES. (Die syringomyelischen (437) Bulbärerkrankungen.) O. P. GERBER, *Wien. med. Woch.*, Aug. 24, S. 1674.

Two interesting cases are here reported.

Case I. Cervical syringomyelia with total anæsthesia of the left cornea. The patient, a woman of 43, had suffered for 22 years from typical symptoms of syringomyelia. Five years after the onset of these there began nasal regurgitation of food and alteration in phonation. Examination showed characteristic signs of cervical syringomyelia, with no addition; nystagmoid tremors, incomplete loss of sensation to pain and temperature in the region of the left trigeminus—tactile æsthesia remaining normal. Complete loss of left corneal reflex and left pharyngeal reflex; the tongue deviated to the right on protrusion. It is curious how the upward extension of the disease had apparently occurred only on the left.

Case II. Humero-scapular syringomyelia, with apoplectic attacks of severe dyspnœa and tachycardia. The patient, a man of 23, had been well till three years previously. Attacks occasionally occurred in which feelings of suffocation and as if the heart were stopping were very prominent; consciousness was thrice lost, and the diagnosis of hysteria was made in the hospital. Advanced atrophy of the humero-scapular muscles was present; the exact state of sensation could not be determined on account of the patient's lack of intelligence. Nystagmoid movements in the upward direction, diminution of the left corneal reflex, protrusion of the tongue towards the right, tachycardia, weakness of left sterno-mastoid and trapezius were the signs pointing to implication of the cranial nerves. ERNEST JONES.

STUDIES ON TABES, WITH ESPECIAL CONSIDERATION OF THE
(438) **LARYNGEAL SYMPTOMS.** [**Studien über Tabes dorsalis mit besonderer Berücksichtigung der Kehlkopfsymptome (221 Fälle).**] GRAEFFNER, *Münch. med. Woch.*, Sept. 3, S. 1775.

THIS valuable paper must be read in full, for it is a condensation of a special study of a large number of late cases undertaken with the view of establishing the relative frequency of the various laryngeal symptoms of tabes. Amongst the conclusions may be mentioned the following. Hyperæsthesia was found in 1·7 per cent., hypo-æsthesia in 13·2 per cent., and complete anæsthesia not once. The author mentions the recent discussions of the Semon-Rosenbach law on the motor side, and supports it strongly. Laryngeal paralyses were present in 26·2 per cent., and laryngeal crises in 13 per cent. Twenty per cent. of cases that shewed laryngeal affection at the first examination did so at the second. Syphilitic changes in the larynx were especially common. ERNEST JONES.

THE EARLY RECOGNITION OF TABES DORSALIS. CHARLES
(439) ROSENHECK, *Med. Record*, Aug. 31, 1907, p. 355.

THIS paper is simply a short account of the symptoms and signs that may be found in the pre-ataxic stage, from which a diagnosis may be made before all the classical symptoms appear.
J. H. HARVEY PIRIE.

WORD AMNESIA AND THE LEFT INFERIOR PARIETAL
(440) **LOBULE.** (**Amnesia verbale e lobulo parietale inferiore sinistro.**) VEDRANI, *Rivista di Psicologia applicata alla Psicopatologia*, Anno iii., pp. 31 and 116.

VEDRANI in an article of 29 pages gives a good review of the published cases and views held on the subject of word amnesia. He reaches no new conclusions, but insists on the importance of the left inferior parietal lobule in relation to language.

He relates the case of a woman aged 80 who presented no physical signs beyond weakness of the lower extremities. She had agraphia, word-blindness, but no word-deafness. The disturbances of speech were profound. She manifested paraphasia, perseveration, jargonaphasia, verbigeration, logorrhœa, impossibility of repeating words uttered, and above all absolute and constant word amnesia. At the autopsy was found an area of thrombotic softening affecting the supramarginal gyrus. The first temporal convolution was slightly implicated, especially as regards its grey matter. ERNEST JONES

A CASE OF LEFT-SIDED APRAXIA AND AGRAPHIA. (**Ein** (441) **Fall von linksseitiger Apraxie und Agraphie.**) OTTO MAAS, *Neurolog. Centralbl.*, Sept. 1, p. 789.

MAAS relates an interesting case which not only belongs to the small category in which left-sided dyspraxia was unaccompanied by paralysis or dyspraxia of the right hand, but was also peculiar in that the lesion was probably in the left hemisphere, not in the corpus callosum.

The patient, a right-handed man, had suffered from a stroke six months previously, which was followed by right-sided hemiplegia and speech disturbance. The hemiplegia, however, cleared up, and at the time in question there was no trace of it or of any aphasic symptom. The patient could write naturally with the right hand, but with the left could write neither spontaneously nor to dictation. This complete incapacity contrasted with his power to copy handwriting with the left hand. All movements with the left hand and arm were grossly dyspraxic, those with the right being normal.

Maas considers that there was a vascular lesion situated at a position, which he illustrates diagrammatically, where the fibres leaving the left hemisphere for the corpus callosum part from the projection fibres of the pyramidal system.

ERNEST JONES.

CASE OF ALEXIA AND AGRAPHIA FOLLOWING TRAUMA. (442) (**Ein Fall von Alexie und Agraphie nach Trauma.**) ALESSANDRO MARINA, *Neurolog. Centralbl.*, Okt. 1, S. 882-894.

MARINA relates the following interesting case. Ths patient, a man aged 21, had suffered from epileptic attacks for a month when three years old. After this he had good health till, when seventeen years old, he fell five metres on his head and was picked up unconscious. He remained unconscious for a week, and on coming to had motor aphasia, alexia, and agraphia; these were absolute, he could neither speak, read, nor write a single word. The aphasia disappeared within a year, but three years after the fall he developed fits. These were typically epileptic in character, occurred more often at night, and were very frequent, often recurring several times a day; there were never any Jacksonian manifestations. In addition, he suffered from attacks of a different kind, which lasted an hour or more; in these he was very excited, complained of stabbing pains in the tongue and face, with feelings of strangling in the throat and neck, lost his voice, and was compelled to drink glass after glass of water. His sleep was restless and disturbed by

nightmares. He was readily tired and could do no work. He had feelings of intense anguish, with fear of impending fits, of death, etc.

On examination was found diminished and retarded pharyngeal reflex, abnormal widening of the visual field for green, slight hypo-æsthesia on the right half of the body, slight exophthalmos and paresis of convergence; no other signs of Basedow's disease were present. The alexia was almost absolute. The patient could read no words except his own name, and only a few letters. He could read figures, however, and recognised all pictures and colours absolutely. The agraphia was equally pronounced. He could write any figures either spontaneously or to dictation, could draw objects passably well, but could write only a few letters to dictation —no words. He could laboriously copy words as if drawing a strange object.

Marina discusses whether the above symptoms were attributable to the traumatic hysteria, which was obviously present, or to an organic cerebral lesion. Against the presence of a lesion in the occipital lobe speaks the absence of visual disturbance; against one in Wernicke's area is the absence of word deafness; against one in the parietal lobe is the perfect stereognosis; and against one in the posterior part of the frontal lobe is the absence of any apraxia. The fact that the complete motor aphasia disappeared entirely at the end of a year points strongly to a functional disturbance, Charcot's "aphasie sans mot des hystériques." The alexia was of the so-called pure subcortical type, yet the patient could read his name and figures, and could carry out simple arithmetical operations. It was noteworthy that when his Christian name alone was written he could not read it until the surname was added, and further that he could not read much simpler Christian names than his own. The agraphia was intimately allied with the alexia; what he could read he could write, and vice versa. When he copied handwriting he changed certain letters, particularly "r," so that the process was not the mechanical one it seemed to be; in other words, his subconsciousness appreciated that it was an "r" he was writing, although he could not read it. The absence of the hemianopsia which occurs in most cases of alexia of organic origin; the capacity to carry out arithmetical operations, which Von Monakow says is always lost in such cases, even if the capacity to read *certain* letters and words is retained; the fact that the alexia was a specific memory defect and not dependent on lost conceptions of the words in question as it is in organic cases; the fact that no sure evidence of organic disease was present, whereas hysteria certainly was, all impel Marina to the conclusion that the alexia, and the agraphia secondary thereto, were of a hysterical nature. The second variety of fits he considers to be also hysterical, but he calls the first variety epileptic, probably from ignorance of the fact that true epileptiform fits may sometimes

occur in hysteria. It is unfortunate that no mental analysis, even of the dreams, was made, for evidently it would have been invaluable for both diagnostic and therapeutic purposes. ERNEST JONES.

A CASE OF TUMOUR OF THE CORPORA QUADRIGEMINA.
(443) CARLO RIGHETTI, *Riv. di Patol. nerv.*, Vol. xii., F. 5.

THE author describes a case of tumour of the corpora quadrigemina studied in a boy aged twelve.

The principal clinical features were—A prominence of the left frontal eminence. Slight exophthalmus on the left side with no disturbance of the ocular movements. Complete loss of sight in the left eye, and well marked optic neuritis in both eyes. Left-sided anosmia. An ataxic gait due to inco-ordination of the muscles on the left side. Loss of memory and a mischievous perversion of the character. "Cracked pot" sound on percussion of the cranium. A diagnosis of a neoplasm of the left frontal lobe was made, and the patient was operated on. He died immediately after an unsuccessful attempt to find the tumour. Post-mortem a diffuse glioma of the quadrigeminal bodies with marked secondary hydrocephalus was discovered. The aqueduct of Sylvius was completely obliterated. On microscopical examination the tumour was found to be composed of small cells rich in chromatin material and with oval nuclei. The cells were grouped in collections of four and five, and surrounded by fine fibrillæ. The ependymal epithelium did not terminate abruptly in the vicinity of the neoplasm, but was stratified, the cells becoming, as the neoplasmic tissue was approached, more and more difficult to distinguish from the tumour cells. Small cavities were found in the tumour lined by ependymal epithelium which also merged insensibly into the glia elements. Such ependymal-lined cavities were never found isolated, but traces of an ependymal proliferation might always be found leading from the ependyma to their vicinity. From the appearance of these cysts the author concludes that the neoplasm arose from cystic ependymal rests developed during the closure of the primitive neurenteric canal. F. GOLLA.

THE HÆMORRHAGIC FORM OF TUBERCULOUS MENINGITIS IN THE ADULT. (La forme hémorragique de la méningite tuberculeuse de l'adulte.)
(444) PAUPE, *Thèse de Paris*, 1906.

HÆMORRHAGE, which is a common process in all forms of tuberculosis, may occasionally occur in tuberculous meningitis. The vascular lesions of endo- and periarteritis are the same as those met with in pulmonary tuberculosis, and the phenomenon may

therefore be called meningeal hæmoptysis. The symptoms are indefinite, and diagnosis can only be made by lumbar puncture, when almost pure blood escapes. This can be distinguished from an accidental hæmorrhage caused by the syringe, in which case only a very slight amount of blood would flow out. Cytological examination as a rule shows a lymphocytosis, which is either pure, or is associated with polynuclears and endothelial cells. In one case, however, reported by Paupe, it was impossible to detect the slightest leucocytic reaction. J. D. ROLLESTON.

THE BABINSKI PHENOMENON. F. PETRO, *Annali di Freniatria*, (445) May 1907.

THE author gives a fairly complete review of modern work on the Babinski reflex with a valuable bibliography, and alludes briefly to his own researches. The observations were made on patients in the central lunatic asylum of Como.

In every case the phalangeal articulations were examined to exclude fixation of the joints from old arthritis, the patient was stretched on the bed with the knee semiflexed and everted, and the attention was as far as possible diverted. The reflex was obtained with the pointed end of a percussion hammer, or, in cases in which it was very depressed, by stroking the foot with a pin. Seven hundred patients were examined. In thirty-three per cent. of the positive cases the patients were suffering from general paralysis, and the majority of these cases showed other signs of disturbance of the pyramidal tracts.

Epileptic patients were found to give a positive response both during and after fits, and equally before them. This statement is so at variance with general experience that it could be wished that some more details had been given concerning the condition of patients giving an extensor response when examined before fits. In twenty-five cases of Pellagra the extensor response was obtained in eight per cent. In one case of dementia præcox, and in another of amentia, the extensor response was obtained. In all the cases which gave the Babinski phenomenon there was increase of the deep reflexes. F. GOLLA.

THE PHYSIOLOGY OF THE KNEE-JERK. U. SCHEVEN, *Pflugers* (446) *Archiv* 117, p. 108.

ACCURATE time measurements show that the latent period between a momentary percussion of the tendon and the quadriceps response is longer than that for direct muscle stimulation by a single induction shock, and that the difference is considerable. This

result would appear to afford some ground for the view that the knee-jerk is a true reflex.

If the tendon is stimulated with stimuli of constant strength and medium intensity while the frequency is increased, the responses are found to increase with the diminution of the period between successive stimulations. Such a result can only be explained as a summation of stimuli. The author considers that the occurrence of this summation is very strong evidence for the true reflex nature of the knee-jerk, the frequently succeeding stimuli causing an increased excitability of the reflex centres.

F. GOLLA.

TWO CASES OF ACUTE TUBERCULOUS CEREBRO-SPINAL (447) **MENINGITIS.** WILSON and MILLER, *Lancet*, Sept. 14, 1907.

THE first case was that of a servant girl aged 17 years, whose illness began with sore throat, vomiting, pains in back and limbs, and other symptoms characteristic of influenza. Thirteen days later symptoms of meningitis developed, and subsequently both legs became completely paralysed. On the eighteenth day a rapid extension of the paralysis to the arms and respiratory muscles resulted in death from asphyxia. On post-mortem examination the spinal pia-arachnoid was found to be opaque and grey in colour and adherent to the cord by means of a sticky exudate. The nerve roots were thickened, and the cord, especially in the upper cervical region, was soft and almost fluid. The thickening of the pia-arachnoid was due to the presence of large numbers of cells which in places were arranged to form typical tubercle follicles. Tubercle bacilli were present but not numerous. There was considerable turbid exudate in the subarachnoid space at the base of the brain, extending over the cerebellum and up the Rolandic fissure, but no distinct tubercles were to be seen. The second patient was a boy of 7 years, who was admitted to hospital with a history of frontal headache and vomiting which had lasted seven days. On the fourteenth day of his illness, twitching of the left side, followed by unconsciousness, dilatation of pupils, Kernig's sign, and rigidity of the neck developed. Double otitis media was present, but trephining on the right side revealed nothing but increased tension, and the patient died next day. At the necropsy a tuberculous bronchial gland was found on the left side. On opening the spinal dura, turbid fluid escaped, and the pia-arachnoid was pale and thickened, but no tubercles were visible. Microscopic examination, however, decided the tuberculous nature of the condition. There was turbid exudate at the base of the brain, extending up the Rolandic fissures and over the vertex. The ventricles were distended with turbid blood-stained fluid.

HENRY J. DUNBAR.

NEURASTHENIA AND GENERAL PARALYSIS. (Neurastenia e
(448) **paralisi progressiva.)** P. PETTRAZANI (of Reggio nell' Emilia), *Riv. sperim. di Fren.*, Vol. xxxiii., Fasc. 2, 3.

IN a long and somewhat discursive article, followed by a large bibliography, the author maintains that the neurasthenic syndrome which frequently precedes the full development of general paralysis is to be considered a true neurasthenia which later gives rise to a general paralysis, rather than merely the expression of a process of general paralysis already existing.

He takes seriatim the symptoms of true neurasthenia as described by the various authors, and shows that they are the same as the early symptoms of what is called the neurasthenic period of general paralysis; he draws a similar parallel between the ætiology of the two conditions, but admits that while syphilis plays a preponderant rôle in the genesis of general paralysis, it is of no special importance in neurasthenia. He sums up by saying that the condition of chronic auto-intoxication, which gives rise to the clinical picture of neurasthenia, may later, in a nervous system prepared by syphilis, give rise to general paralysis.

C. MACFIE CAMPBELL.

ON HEREDITY IN GENERAL PARALYSIS. (Sull' influenza dell'
(449) **ereditarieta morbosa nella paralisi progressiva.)** G. FORNACA (of Rome), *Riv. sperim. di Fren.*, Vol. xxxiii., Fasc. 2, 3.

A STATISTICAL contribution to the question of the importance of heredity as an ætiological factor in general paralysis, with the genealogical tables of twenty-four patients.

C. MACFIE CAMPBELL.

RECENT THERAPEUTIC ATTEMPTS IN GENERAL PARALYSIS.
(450) **(Recenti tentativi di cura della demenza paralitica.)** B. G. S. ESTENSE, *Riv. sperim. di Fren.*, Vol. xxxiii., Fasc. 2, 3.

A CRITICAL review of the experiments made with anti-syphilitic treatment, saline infusions, injections of tuberculin, of artificial serum, of serum of paralytics in remission (Bruce), of vaccines and specific anti-sera (Robertson and M'Rae).

C. MACFIE CAMPBELL.

A NEW FORM OF SYNÆSTHESIA. (Una nuova varietà di
(451) **sinestesia.)** FERRARI, *Rivista di Psicologia applicata alla Psicopatologia*, Settembre-Ottobre 1907, Anno iii., p. 297.

FERRARI relates the following interesting case. The patient, a woman of 25, experienced the savour of different foods when

different proper names were suggested to her, and with this the corresponding pleasure or distaste appropriate to the given food. She was tested repeatedly with a list of 200 names; 163 of these constantly gave rise to the image of a certain food; with the other 37 this was less definite. The observations lasted over many months and the results were remarkably constant; in only a few cases did the answers differ. The same image might be evoked by any one of several names in a couple of instances. On the other hand the thought or sight of a food did not call up the corresponding name. The association occurred much better when the names were uttered aloud than when she read them; and it was interesting to note that several names with which she was very familiar in literature but which she rarely heard mentioned gave rise to no such association. The patient had experienced this phenomenon as long as she could remember; no other abnormal symptoms were present.

Ferrari studied the nature of the associations, finding often a phonetic connection. He makes some valuable remarks on the theory and pathogenesis of the condition. ERNEST JONES.

SYNERGIC MOVEMENTS OF THE EYELIDS AND MOUTH. (452) W. M. BEAUMONT, *Brit. Med. Journ.*, Sept. 14, 1907.

A CASE is recorded of congenital ptosis with epicanthus in a child. Opening of the mouth is produced by any effort to raise the eyelids.

Such associated movements of eye and mouth may frequently be observed under pathological conditions in children. Nor are they only seen in pathological cases. Opening of the eyes is found normally to cause relaxation of the closing muscles of the jaws. The open mouth is present together with the widely open eyes in gaping.

An analysis of recorded cases of associated eye and mouth movements shows that they are characteristic of the young or of those who have not acquired complete control over their facial muscles. They tend to disappear as development progresses. The author, who quotes Darwin's statement that the young show less modification and more resemble ancestral types than adults, considers that we have here vestigial traces of association of movements which characterised ancestral forms. The marked appearance of mouth movements in ptosis, when an attempt is made to raise the eyelids, is due to overflow affecting the mouth muscles from the abnormally exaggerated impulse to the eyelids. W. A. JOLLY.

PATHOLOGY OF PARALYSIS AGITANS. (453) C. CAMP (of Philadelphia), *Journ. of the Am. Med. Ass.*, April 13, 1907.

A BRIEF summary of recent literature on the subject is made; several of the theories as to the nervous origin of the disease are

discussed; case reports are given. The author's conclusions are as follows:—

1. Paralysis Agitans is not a neurosis, nor is it senility.
2. The anatomical basis of the muscular rigidity, tremor, and the symptoms dependent upon them, lies in the affection of the muscles.
3. The disease itself is probably a toxæmia, and there is suggestive evidence that it is due to alteration in the secretion of the parathyroid glands. C. H. HOLMES.

EPILEPSY AND LIKE CONDITIONS IN CHILDHOOD. (Ueber
(454) **Epilepsie und epileptoide Zustände im Kindesalter.)** GUSTAV ASCHAFFENBURG, *Arch. f. Kinderheilk.*, Bd. xxvi., H. 3-6.

THE author describes epilepsy as "periodic variations of the psychic equilibrium, which lead, according to the more or less general involvement of the central nervous system, to slight or severe interruptions of consciousness, and which may be, but are not necessarily, accompanied by convulsive seizures." Pædiatric physicians have this, he considers, to learn from psychiatric physicians—namely, that interruptions of consciousness and not convulsions are characteristic of epilepsy. A. DINGWALL FORDYCE.

IS EYE-STRAIN EVER AN ETIOLOGICAL FACTOR IN EPILEPSY.
(455) H. REIK (of Baltimore), *Journ. of the Am. Med. Ass.*, May 4, 1907.

THE author reports four cases of epilepsy in which the active causative factor appeared to have been eye-strain; the correction of the refraction errors appeared to relieve the greater affection. His own observations have convinced him that epileptic seizures may be induced by reflex action from ocular disease. It has been estimated that there is one epileptic to every 500 inhabitants in this country; less than one per cent. are ever restored to health. If, then, only one case in a hundred could be benefited by this mode of treatment, the percentage of cures would be doubled. The removal of a reflex excitant does not of course cure the disease; the predisposition remains, and if the patient is again subjected to eye-strain the attacks will probably recur. C. H. HOLMES.

TACHYCARDIA OF BULBAR ORIGIN IN TYPHOID FEVER.
(456) **(La tachycardie. Origine bulbaire dans la fièvre typhoïde.)** TCHILIAN, *Thèse de Paris*, 1905.

TCHILIAN thinks that the frequency of myocarditis in typhoid fever has been considerably exaggerated. The tachycardia ob-

served in the course of that disease is most often the result of the action of the typhoidal toxines on the medulla. Naked eye and microscopic examination in such cases has often failed to find any morbid changes in the heart. Tachycardia may be present at the onset of typhoid, or develop at a late stage of the disease. It may occur as an isolated symptom, or be associated with ataxo-adynamic phenomena. In the 16 cases reported by Tchilian, tachycardia was associated with characteristic signs of bulbar involvement, such as cardiac asthenia, weakness of the pulse, irregularity of the heart sounds, Cheyne-Stokes rhythm, cyanosis, dysphagia, hiccough, dyspnœa without any pulmonary lesions, and vomiting.

The prognosis is grave if the pulse rate is 140 or over, but may be regarded as relatively favourable if the pulse is not above 130.

J. D. ROLLESTON.

TWO CASES OF HEREDITARY DYSTROPHY. W. OGILVY and (457) P. G. EASTON, *Brit. Med. Journ.*, Oct. 5, p. 867.

TWO cases of dystrophy occurring in father and son are here published. The sterno-mastoid, pectorales, deltoid, trapezius, spinati, serratus magnus, rhomboidei, latissimus dorsi and erector spinae were the muscles affected. Lordosis and scapula winging were pronounced. The electrical reactions were not tested. In both cases the affection began about the age of 23. The son's son, aged 3, and grandfather were normal; no other relatives are mentioned.

ERNEST JONES.

ACUTE INFANTILE POLYNEURITIS; INFANTILE SPINAL (458) **PSEUDO-PARALYSIS.** LÉON, *Nouvelle Iconographie de la Salpêtrière*, May-June 1907, p. 216.

ACUTE infantile polyneuritis may begin in an insidious way, which may serve to distinguish it from the onset, usually more or less abrupt, of poliomyelitis anterior acuta: this distinction, however does not always obtain. Pains, spontaneous or provoked by change of position, or elicited by pressure on muscles or nerve trunks, are of considerable diagnostic significance, but they are not obvious in the motor forms: on the other hand, they occasionally occur in anterior poliomyelitis.

The intense pain derived from faradic stimulation of nerve trunks is not of the same importance that it is in adults, since children do not bear electrical examination well. The cranial nerves are frequently involved in the paralysis of polyneuritis, which, further, is of greater intensity peripherally than centrally. The type of regression in the two diseases is commonly different.

In polyneuritis the sensory and motor topography is peripheral rather than radicular. One can usually come to a definite diagnosis by means of a study of the electrical reactions.

S. A. K. WILSON.

(459) **PSEUDO-HYPERTROPHIC MYOPATHY OF THE CALF AND SHOULDER GIRDLE MUSCLES; WITH ATROPHY OF THE PECTORALIS MAJOR.** RAYMOND and ROSE, *Nouvelle Iconographie de la Salpêtrière*, May-June 1907, p. 224.

A BOY of ten years presents the aspect of the calf muscles which is characteristic of classical pseudo-hypertrophic muscular paralysis: in addition there is an unusual hypertrophy of the deltoids, subscapulares, and serrati magni, coupled with great atrophy of the pectoralis major. The oblique abdominal muscles also are enlarged, and so are the right vastus externus and the left glutei. Fibrillation is conspicuous by its absence, and no reaction of degeneration is obtainable.

The case seems to be a combination of the pseudo-hypertrophic type of Duchenne with Zimmerlin's type (atrophy of the pectoralis major), and the juvenile type of Erb.

S. A. K. WILSON.

(460) **THE INFLUENCE OF ANTITOXIN UPON POST-DIPHTHERITIC PARALYSIS.** ROSENAU and ANDERSON, *Bulletin No.* 38, *U.S. Public Health and Marine-Hospital Service*, June 1907.

THE experiments conducted by Rosenau and Anderson prove that post-diphtheritic paralysis in the guinea pig is almost exactly similar to that occurring in man. Guinea pigs may develop paralysis after the injection of a sublethal dose of toxine, or the injection of a toxine-antitoxin mixture in which the toxine is but partially neutralised. By no means every guinea pig that develops paralysis dies. Recovery is frequent, and depends entirely upon the amount of toxine in the toxine-antitoxine mixture. It is very easy to mix the toxine and antitoxine in such proportion that every guinea pig will develop post-diphtheritic paralysis and die. By adding a little more antitoxine, or a little less toxine to the mixture, the animals will develop mild paralysis, from which almost all recover. Paralysis in the guinea pig can rarely be detected before the 14th day, and is manifested by a softness of the abdominal muscles near the site of injection. The weakness gradually spreads to the extremities, and finally to the muscles of respiration. Early paralysis is almost always fatal, late symptoms are usually mild, and end in complete recovery. The paralysis

may be slight, affecting one limb only, or slightly embarrassing respiration, or it may be severe and cause sudden or gradual death. It may last for two days or two weeks, or it may terminate fatally at any time. It is never permanent. To settle the question whether antitoxin can influence diphtheritic paralysis after the paralysis has appeared, the writers injected guinea pigs with 400 or 500 units of antitoxin during various stages of the paralysis. They found that antitoxin so administered had no power to save life or ameliorate the symptoms. Neither had antitoxin any influence in preventing paralysis if injected shortly before the paralysis develops. On the other hand, a very small quantity of antitoxin given 24 hours before, or at the time of infection, prevented the development of paralysis, and antitoxin given 24 hours after the infection saved the life of the guinea pigs and greatly modified the paralysis. A single large dose given 48 hours after infection did not modify the paralysis or save life. Repeated injections, however, given 24-48 hours after infection seemed to have a more favourable influence upon the subsequent paralysis than a single injection. In conclusion the writers state that more favourable results might be expected from the use of antitoxin in man than their work on the guinea pig indicates, in which they were dealing with an early and malignant form of experimental post-diphtheritic paralysis. This grave variety is rare in man. Further, the entire charge of the poison was injected directly into the tissues of the guinea pig, while in man the toxine is doubtless elaborated more slowly. They, therefore, assume that antitoxin given at a somewhat later period than in the case of guinea pigs would exert beneficial effects.

(This assumption is verified by clinical experience. In 198 cases of diphtheria under the reviewer's care, in which antitoxin was first injected after the fifth day, the mortality was 12.1 per cent., whereas in pre-antitoxin days the mortality of diphtheria was never less than 28 per cent. *Cf. Lancet*, 1, 1907, p. 1112).

J. D. ROLLESTON.

HYSTERICAL MYDRIASIS. LAFON and TEULIÈRES, *Nouvelle Icono-*
(461) *graphie de la Salpêtrière*, May-June 1907, p. 243.

THE patient was a man, aged 34, who had never noticed anything wrong with his vision, though he seems to have been aware of the fact that his right pupil was larger than its fellow. He was unable to say when he had first remarked this difference.

The right pupil was in a state of maximal mydriasis, inactive to light, on convergence, to painful stimuli; further, the consensual reflex was awanting. The left pupil was of medium size, and reacted sluggishly to light through a small range: this con-

traction was not sustained. Its reaction on accommodation was slight, and it did not change with pain. Corneal and conjunctival reflexes and sensibility were normal. There were six dioptres of myopia in the right eye, and eight in the left. Corrected vision was 6/12 in the right eye, and 6/18 in the left. There was a slight concentric diminution of the fields for white, but no dyschromatopsia. A slight external strabismus of the left eye also existed. The only other point to be noted was a pronounced left hemi-hypæsthesia.

A drop of cold water dropped into the right eye produced an instantaneous and energetic contraction of the iris, and after about a minute it gradually returned to its former condition. A drop of ½ % solution of eserine caused at first an energetic contraction of the pupil, which returned to its usual position in about a minute: after about six minutes an intense myosis was gradually produced; the patient now said he saw indistinctly, everything being tinted green. The contracture of the iris and the ciliary muscle eventually relaxed in the ordinary way.

One drop of a 4 % solution of cocaine produced the same phenomena as a drop of cold water: the mydriasis was not subsequently influenced. One drop of ½ % solution of atropine, after the usual instantaneous contraction, was followed by a paralysis of accommodation which persisted for eight days. The pupil reacted to an alternating current, the positive pole being on the back, the negative on the closed eyelid. In the left eye, cold water produced no effect, and its reactions to these various solutions was normal.

The authors are satisfied that the mydriasis does not conform completely to the characters either of the paralytic or the spasmodic type. They consider it is the result of a lesion in the ciliary ganglion or in the nerves which leave that body. There cannot be an organic paralysis of the sphincter, nor can the error of refraction account for the mydriasis. A professional cramp may be thought of, since the patient is a watchmaker, but that hypothesis clearly cannot be entertained. A functional paralysis of the sphincter of the iris seems indicated.

S. A. K. WILSON.

THE NEURASTHENIC NEURALGIAS. F. K. HALLOCK, *New York* (462) *Med. Journ.*, Sept. 7, 1907, p. 442.

THIS paper deals with a group of neuralgias symptomatic of an underlying neurasthenia, and clinically lying between indefinite subjunctive paræsthesia and definite objective neuritis. The symptomatology thus varies very extensively, but it is almost pathognomonic that the patient exhibits the pain more in his

description than in his appearance. Many cases have well-marked local symptoms and, the writer states, are diagnosed as neuritis, but the fluctuation of the symptoms with the psychic state of the individual makes it impossible to reconcile this diagnosis with our conception of an actual inflammation of a nerve trunk. Treatment is usually satisfactory, but it must be directed mainly towards the fundamental neurasthenia; local treatment is not of much service further than rest at first, with more active treatment in the later stages of recovery. It is particularly unwise to label the condition "neuritis" or to make the symptoms too objective, as this tends to fix the idea in the patient's mind and lead to a long continuation of the symptom. J. H. HARVEY PIRIE.

THE PATHOLOGY OF DIVERS' PALSY. (Les lesions anatomo-
(463) **pathologiques de la moelle épinière, etc.)** ZOGRAFIDI, *Nouvelle Iconographie de la Salpêtrière*, May-June 1907, p. 208.

IN six cases of divers' palsy, where death occurred at varying intervals after the attack, the author found indications of myelitis in the spinal cord. In two instances the myelitis was intense; hæmorrhagic foci existed in four; in all were diffuse or circumscribed necrotic areas corresponding to the distribution of the spinal arteries, with wide spaces where altered material had been absorbed. In one case, where the patient died on the fifth day, in addition to the first stage of myelitis as exemplified by congestion, leucocytic diapedesis, swelling of nerve fibres, etc., there were large dilatations or aneurisms of the vessels themselves. In cases which are not immediately fatal the clinical picture becomes one of chronic spastic paraplegia, with, pathologically, sclerosed areas in the lateral and posterior columns of the cord.

S. A. K. WILSON.

PSYCHIATRY.

REMARKS ON PROF. SPECHT'S PAPER ON "ANGSTAFFEKT"
(464) **IN MANIC-DEPRESSIVE INSANITY. (Bemerkungen zu dem Aufsatze von Prof. G. Specht: Ueber den Angstaffekt im manisch-depressiven Irresein.)** A. WESTPHAL and O. KÖLPIN, *Centralbl. f. Nervenheilk. u. Psychiat.*, Okt. 1, S. 729.

THIS is a short criticism of a few of the points raised by Specht in his recent remarkable essay on this subject.

Specht concludes that Wernicke's *Angstpsychose*, which he identifies with Melancholia agitata, is a mixed form of manic-

depressive insanity, on the grounds that flight of ideas (Ideenflucht) and impulsive speech (Rededrang) occur therein. Wernicke, however, defined Melancholia agitata as only a special form of *Angstpsychose*, its characteristic being persistent motor restlessness, while flight of ideas and impulsive speech are only facultative manifestations thereof. Westphal and Kölpin deny further that Specht is justified in regarding forced movement (Bewegungsdrang) and melancholic flight of ideas as manic manifestations; they consider these are direct manifestations of the Angst itself. They therefore deny Specht's conclusion that *Angstpsychose* is always to be regarded as a mixed form of manic-depressive insanity, though they grant that he is justified in so placing those varieties of *Angstpsychose* that exhibit true manic symptoms, such as pure flight of ideas, distractability, etc.

They agree with Specht that senile melancholia cannot be separated from the whole manic-depressive group (a view that is gradually gaining ground in German psychiatry).

ERNEST JONES.

ON PSYCHOPHYSICAL RELATIONS OF THE ASSOCIATION
(465) **EXPERIMENT.** C. JUNG (of Zürich), *Journ. of Abn. Psych.*, Feb. 1907.

THE author conducts an electric current of about two volts through the human body by means of poles applied to the palms. A galvanometer of high sensibility is introduced into the circuit, and a careful watch is kept upon the galvanometer as tactile, optic, or acoustic irritations of a certain strength are applied to the subject. The author finds that commonly the galvanometer shows an increase in the amount of the current, *i.e.* a lowering of the electrical resistance of the body. It was noted that the action of the galvanometer was often in direct relation to the intensity of the resulting psychical feeling-tone, rather than to the strength of the irritation; further, the inconstancy of the galvanometer often did not occur until from one to six seconds after the perception of the irritation. Veraguth found that often the mere announcement of irritation caused a marked response, and concluded that feelings were objectively represented in the experiment.

Jung has previously shown that on performing association tests strong feeling-tones often accompany the associations, causing regular and characteristic disturbances in the association process. By plotting curves of the galvanometer oscillations during association experiments he obtained some very interesting results, and suggests that the method may be a suitable way of demonstrating

the feeling-tones which accompany associations. He admits fairly that the experiment possesses many complications which time and work will alone overcome. C. H. HOLMES.

PSYCHO-PHYSICAL INVESTIGATIONS WITH THE GALVAN-
(466) **OMETER AND PNEUMOGRAPH IN NORMAL AND INSANE INDIVIDUALS.** F. PETERSON (of New York) and C. G. JUNG (of Zürich), *Brain*, July 1907.

IN this communication the authors give, in some detail, their observations made to ascertain the value of the "psycho-physical galvanic reflex" as a recorder of psychical changes in connection with sensory and psychical stimuli. Their desire to find an objective record of such mental phenomena led them to include in their research the study of the respiratory curve as an expression of emotion. In the curve obtained by the pneumograph, as in the curve obtained by the galvanometer, emotional variations are recorded, but between the two curves there is often very marked divergence.

No general conclusions are reached, and the authors make the communication more to stimulate research than to publish valuable results. C. MACFIE CAMPBELL.

ALCOHOL AND INSANITY—THE EFFECTS OF ALCOHOL ON
(467) **THE BODY AND MIND AS SHOWN BY ASYLUM AND HOSPITAL EXPERIENCE IN THE WARDS AND POST-MORTEM ROOM.** F. W. MOTT, *Arch. of Neurol.*, Vol. iii. 1907.

THIS is a paper which, in the course of 57 pages, deals with manifold aspects of alcoholism. There are statistics of its frequency as an ascribed cause of disease at Charing Cross and St George's Hospitals, and at the several London County Council Asylums. Etiological relationships are touched upon with regard to age, sex, occupation, and hereditary and social influences. There is a section dealing with the effects of alcohol in producing cirrhosis of the liver, with a further series of statistics from Charing Cross and St George's Hospitals, and from Claybury Asylum; and the writer draws attention to the infrequency of cirrhosis of the liver among the insane. There are lists of the diseases which may be directly ascribed to the abuse of alcohol, and of those of which alcohol may be considered a contributory cause.

Coming to the asylum cases in which alcohol was set down as the ascribed cause of the mental disorder, Dr Mott again dis-

tinguishes between those of which alcohol is merely a contributory cause and those of which it is the essential cause, the latter class (probably less than ten per cent. of the total admissions) comprising those patients who would never have seen the inside of an asylum but for alcohol.

Apropos of the latter class, reference is made to some of those symptoms which, taken in conjunction with others, are characteristic of an alcoholic psychosis; such are hallucinations, loss of memory, boastful loquacity, untruthfulness, and especially fabrication, with loss of orientation in time and place.

As one of the tests of memory, Dr Mott applies the following, originally devised by Marie for cases of aphasia, to cases of alcoholism:—

"Take three pieces of paper of unequal size. Tell them to "carry out three separate and distinct operations for each piece. "They will be able to carry out each order when separately given "to them, but if before they commence any one, the orders for the "three are given together, they will forget and carry out the orders "imperfectly. Thus, tell the patient to fold up the large piece and "put it in his pocket, the middle-sized piece to be folded and "handed to you, and the small piece thrown on the floor. Whether "it is the lack of power of attention or inability to recollect more "than one order I know not, but the frequency with which failure "occurs in alcoholic subjects shows mental impairment which is not "discovered if any one order is given."

The most interesting part of the paper, however, is an account of the histological changes found post-mortem in cases of the polyneuritic psychosis and of alcoholic pseudo-paresis. In the former condition, Mott finds "some wasting of the tangential fibres and subpial glia cell proliferation and felting, but as a rule this is not marked. The fibre systems are otherwise well preserved. There is no marked glia cell proliferation in the cortex, and when sections are stained by Nissl method the cells of the columns of Meynert are not distorted or poorly stained, their apical processes are not cork-screwed, and there is no marked coarse change of the cortical neurons as in general paralysis. There is no lymphocyte and plasma cell infiltration of the membranes and perivascular sheaths." On the other hand, the large psychomotor cells show characteristic changes, "the nucleus is large and clear, dislocated to the side, sometimes extruded altogether, and there is marked chromatolysis."

The naked eye and microscopic changes in alcoholic pseudo-paresis are also quite unlike those of general paralysis. "Although the membrane may be opaque and thickened, there is but little wasting of the cortex; the floor of the fourth ventricle is not granular, or only slightly so in the lateral sacs. There is microscopically no disorganisation of Meynert's columns, and no

evidence of lymphocytes or plasma-cells in the perivascular lymphatics of the cortex. The only definite microscopic change is some neuroglia cell proliferation in the subpial and septal structures of the cortex, and replacement by it of the association fibres in the tangential and supraradial layers. Generally there is evidence of chromolytic changes of the pyramidal cells and active proliferation of young glia cells.

Incidentally, Dr Mott mentions a severe case of polyneuritis in which a portion of one of the posterior spinal ganglia was destroyed and only a cavity left. "It was the fifth lumbar ganglion, and there was glossy skin on the foot of that side and a trophic sore on the sole." Now we are in the habit of supposing that sensation of the sole of the foot is transmitted by way of the first sacral root. If, therefore, Dr Mott has not made an error in counting the ganglia of this case, this observation means one of two things :— Either Dr Head is one out, all the way down, in the nomenclature of his spinal areas (a question which has been raised before), or else the trophic supply of the sole of the foot is not identical with the sensory supply. This is, of course, quite a side issue, but it is an important one.

It is quite impossible to do justice to the paper in an abstract. There seems to be no aspect of alcoholism which is left untouched; and the paper concludes with sections on Chronic Alcoholism and Legislation, Hanwell Statistics, the Rôle of Alcohol in the Etiology of Epilepsy, and a detailed report of two cases of chronic alcoholism. W. H. B. STODDART.

MORBID SLEEPINESS. C. CAMP (of Philadelphia), *Journ. of Abn.* (468) *Psych.*, April-May 1907.

THERE are two forms of morbid sleepiness—continuous drowsiness and periodical attacks of sleep, varying from mere drowsiness to complete unconsciousness, and lasting from a few minutes to many hours. Of the *continuous* form, which is the most common, there are numerous causes, such as old age, cardio-vascular disease, toxæmias, insolation, cerebral tumours, cranial injuries, etc. (Dana). The *periodical* or second form may be symptomatic of anæmia (Cousins), or auto-intoxication (Caton). It may be associated with hepatic disease (Levi), sarcoma at the base of the brain (Franceschi), neurasthenia (Oppenheim). It may also appear as an epileptic equivalent.

In 1880 Gelineau suggested the term narcolepsy for conditions of this sort.

Camp illustrates the second class of cases by giving a detailed history of a patient who recently came under his notice. Male,

41, strong, apparently healthy, labourer, suddenly begins to have involuntary attacks of sleep many times a day, unaccompanied by physical illness; no hysterical stigmata, slightly heightened irritability, natural sleeping period undisturbed except by visual and unpleasant dreams. Under electricity and suggestion improvement followed in a week's time. The author emphasizes the importance of distinguishing such attacks from petit mal and hysterical somnolence. Oppenheim and Ballet consider narcolepsy as merely a symptom.

Among the theories of sleep he mentions foremost that of Claparède: "It is a positive function, an act of a reflex order and instinct." Camp agrees with this view. The theories of Lorand, who considers sleep as a function of the mind regulated by a secretion of the thyroid gland, and of Salmon, who traces its cause to a secretion of the pituitary body, should be disregarded until more convincingly proven. C. H. HOLMES.

ON CERTAIN CENESTHETIC DISTURBANCES. P. SOLLIER (of (469) Boulogne), *Journ. of Abn. Psych.*, April-May 1907.

IN general the disturbances consist of a sudden sensation of shock, of cerebral cataclysm. It is commonly referred to by the patients themselves as an explosion in the brain, a cracking or bursting sensation, following which they complain of being transformed or changed; the world appears different; their impressions are unreal; a feeling of stupefaction is often followed by one of extreme anxiety; everything seems to escape and vanish from them; the patient is unable to hold on to himself even.

The first patient, a healthy man of 50, actively engrossed in business affairs, experienced a sudden shock in the head; the brain seemed to turn upon itself; immediately he became apathetic and sad. For several weeks he presented the appearance suggested by melancholia, when suddenly he experienced a similar shock and developed extreme mental hyper-activity, which gradually diminished and finally disappeared. Several years later the depressed phase made its appearance in the same fashion, and gradually passed into a state of hyper-activity, which, in turn, disappeared gradually at the end of a year. For the past twelve years the patient has pursued his occupation uninterruptedly, and in a normal fashion.

The second patient was a woman of 40 years, who some time previously had suffered from an attack of melancholia, lasting fifteen months. Gradually she became weary of everything and shunned society. Suddenly she became completely unable to remember things, felt that a veil covered everything, she could not

feel anything, heeded nothing, felt indifferent to everything, and suffered from insomnia. (The outcome of this case is not stated.)

The third patient was an unmarried man of 30. He indulged in onanism in early life and had always believed in mysticism. He was a law student who held an important position. He was subject to frequent headaches and stomach trouble. At night he suddenly awoke, feeling that the circulation of the blood in his brain was being impeded; he became analgesic, was unable to distinguish pain, felt disinterested in everything, and declared himself a living corpse. He has been able to continue his occupation, but in ten years the condition has varied but little.

Jean Jacques Rousseau has described similar sensations which he experienced himself. They are less clear cut than in the others; nevertheless the abruptness, the intensity, and the profound change of feelings are noteworthy.

While circulatory disturbances may be a contributory cause in the development of cenesthetic disorders, the author believes that a permanent change in cortical activity is more fundamental. It is a question whether it is the cortical disturbance which causes the circulatory perturbation, or vice versa. It is to be noted that sensations analogous to those that have been mentioned occur in hysterical patients. Hysteria, in the opinion of the author, is not, properly speaking, a disease, but a state which differs from physiological states only by its intensity and its fixity.

C. H. HOLMES.

A TENTATIVE CLASSIFICATION OF THE PHENOMENA OF
(470) **GLOSSOLALIA. (Essai d'une classification des phénomènes de glossolalie.)** E. LOMBARD (of Neuchâtel), *Arch. de Psych.*, July 1907.

THE phenomenon of glossolalia, or "gift of tongues" of St Paul, is not confined to any age nor to any particular race, nor does it only occur in a religious environment. The author provisionally defines the phenomena under discussion as "vocal automatisms which assume, or tend to assume, the form of a language different from that spoken by the individual in his normal state." With regard to these Flournoy has given the necessary outline of examination: "one must examine if, and how far, the individual attributes a definite meaning to the sounds which he emits; if he understands, or believes that he understands, his own words; or whether his productions are merely the meaningless discharge of the mechanism of phonation, or whether this jargon, unintelligible to the ordinary personality, expresses the ideas of some secondary personality. . . . One may see in the same individual, and sometimes in the course

of the same attack, a series of neologisms, intelligible or unintelligible, followed by simple incoherent verbiage in the ordinary tongue."

As many of the documents bearing on this phenomenon are of considerable antiquity, the author has undertaken a morphological study of the various forms of glossolalia rather than a psychological examination of the conditions under which the phenomenon arises. As a rule, such productions have become the object of mystic interpretation, as they necessarily "appear to the individual or his environment as depending not on the personality of the individual, but on personalities different from and superior to him."

The most elementary form of the phenomenon consists simply in the ejaculation of inarticulate sounds, confused exclamations, cries, murmurs, stammerings. Such ejaculations, which are psychologically a rudimentary glossolalia, are frequently accompanied by a somewhat puerile behaviour, which is due to the breaking through of various childish trends and impulses on the removal of the usual inhibition. The condition under which the phenomenon arises is always one of great emotion, and this often finds its expression in musical forms; the author cites examples from the Old Testament and the recent Welsh revival.

The productions need not be of such an elementary nature, and after the discussion of the above form of glossolalia, a description is given of the more complex forms; the productions here consist of a crude jargon simulating a language, of the introduction of occasional neologisms or of neologisms systematised into a self-coined language, of the introduction of actual foreign words more or less distorted, and finally, of an actual foreign language supposed to be unknown to, or at least unused by, the ordinary personality of the individual.

The whole subject is presented in an extremely interesting and lucid manner. C. MACFIE CAMPBELL.

THE LONGEVITY OF IDIOTS. L. PIERCE CLARK and C. E. ATWOOD, (471) *Med. Record*, Aug. 31, 1907, p. 352.

IT appears to be a common belief that idiotic children rarely live to adult life. The authors have charted the cases in two large institutions for idiots, and their tables show that although life is markedly curtailed, they may, under good care, live well into middle life; thus, of 785 cases in one service, 21 were over 40 years of age. Photographs of some of these twenty-one show that all look much older than they really are; and although idiocy implies an absence of facial expression, they all exhibit a remarkable increase in the number of facial wrinkles J. H. HARVEY PIRIE.

TREATMENT.

THE SANATORIUM TREATMENT OF ACTIVE INSANITY BY (472) REST IN BED IN THE OPEN AIR. C. C. EASTERBROOK, *Journ. Ment. Science*, Oct. 1907; and *Scot. Med. and Surg. Journ.*, Nov. 1907.

THE above papers were read at the annual meeting of the Medico-Psychological Association at London in July, and the International Congress of Psychiatry and Neurology at Amsterdam in September. By active insanity is meant insanity with active symptoms, irrespective of its duration, which may be acute or recent, sub-acute or sub-recent, and chronic or persistent. From 1894 to 1902 the writer practised the time-honoured, and still widely prevalent, "exercise" treatment of active insanity, that is, by regular daily exercise in the open air, and obtained a recovery rate of 42 per cent., the results being slightly better with women than with men. From 1902 to 1906 he carried out the "rest" treatment, that is, by rest in bed indoors on hospital and Weir Mitchell lines, and obtained a recovery rate of 44 per cent., the results being slightly better with men than with women. Apart from this slight improvement in the recovery rate, he found that "rest" possessed other advantages over "exercise" in the treatment of those actively insane, securing for example more satisfactory clinical observation by the physician, better supervision and attention by the nurse, and greater contentment and manageability on the part of the patients. Having seen the marked improvement exhibited by tubercular patients, sane and insane, undergoing open-air treatment, and having noticed also the bodily and mental improvement of the ordinary sick and infirm, but non-tuberculous, insane after an occasional day in the fresh air and sunshine in favourable summer weather, he decided to combine the "rest cure" with the "open-air cure" as a systematic treatment for those actively insane, and made provision for this purpose in 1903 in the design of the new hospital at Ayr Asylum, with the opening of which in 1906 the regular practice was begun. All newly-admitted patients at Ayr Asylum are placed straightway in verandahs attached to the reception wards, and treated with rest in bed in the open air during daylight hours—from 7 A.M. to 7.30 P.M. during most of the year—the course of treatment lasting until the active mental and nervous symptoms subside, and the physical condition, specially the weight, improves. The period of open-air rest lasts one, two, three, or four weeks in the majority of cases, less than a week in the milder cases, and more than a month in cases with continuous active symptoms, these forming the smallest group. Comparing the therapeutical effects of outdoor exercise, indoor rest, and out-

door rest in the subjects of active insanity, the writer has found (1) that outdoor exercise benefits primarily the physical condition, but may retard the mental improvement; (2) that indoor rest benefits primarily the mental condition, but may retard the physical improvement; and (3) that outdoor rest benefits from the first both the mental and the physical condition, and so promotes from the outset both mental and bodily improvement; further, it avoids the risk of undue exhaustion which attaches to the method of exercise, and the evils of sluggish metabolism which attach to prolonged indoor rest. The writer's observations lead him to conclude that the advantages hitherto attributed to "exercise" in the treatment of insanity are due, not to the factor of exercise, but to the concomitant exposure to the open air; and that the disadvantages hitherto attributed to prolonged indoor rest are due, not to the factor of rest, but to the confinement indoors. The open-air rest treatment having proved beneficial in the case of the newly admitted and actively insane patients, the writer then extended its use to the resident and chronic patients during their relapses, the verandahs for these more or less specially noisy and demonstrative cases being subdivided by partitions which isolate the beds from one another, and so prevent one excitable patient from disturbing or being disturbed by others. This experiment similarly proved beneficial, with the result that indoor rest and isolation of actively insane patients during daylight hours has been practically abolished from the institution. As a rule nearly 10 per cent. of the asylum population are being treated with open-air rest for active insanity; and the open-air regime (without rest) during daylight hours has been extended to all others, except meantime, the bed-ridden patients in the infirmary wards. Although it is yet too soon to speak definitely as to the effect of the sanatorium treatment on the recovery rate in insanity, the combination of rest with open air in the writer's experience secures the advantages without the disadvantages of both the rest and the exercise systems as formerly practised, and justifies the expectation that the open-air rest treatment will in course of time yield more satisfactory results than either. AUTHOR'S ABSTRACT.

EFFECT OF EGYPTIAN CLIMATE ON NEURASTHENIA.

(473) (**Wirkung des Klimas Ägyptens auf Neurasthenie.**) GUSTAV HEIM, *Centralbl. f. Nervenheilk. v. Psychiat.*, Okt. 1, S. 732.

BASING his views on the opinions of the most experienced physicians in Egypt, Heim writes in contradiction of the current opinion that that country is a paradise for neurasthenics. In the spring and summer especially, as is shown by the reports of the asylums,

many climatic factors exert an unfavourable influence on mental conditions; particularly is this so in relation to the spring winds and insomnia. He concludes that in Egypt, as everywhere else in the world, climatic influences have no specific effect on neurasthenia; some patients find themselves better at the sea, others in the mountains, some when travelling, others when stationary, etc.

ERNEST JONES.

Review

DIE FUNKTIONEN DES ZENTRALEN NERVENSYSTEMS. M. LEWANDOWSKY. Gustav Fischer, Jena.

IN this handbook the author sets himself the task of presenting a full and complete account of the functions of the central nervous system in portable form. Within its 420 pages it contains a vast amount of material, and one of the outstanding features of the book is the quantity of original and individual work which is embodied in it. Practical facts obtained from the clinique are correlated and compared with the results of experimental research in the laboratory in a manner which is calculated to throw much light on many obscure problems in the physiology and pathology of the central nervous system. Full justice is done to other workers in the same field, the researches of English neurologists and physiologists being duly recognised and appreciated—a statement which cannot always be made with regard to Continental publications. In his treatment of the large mass of facts which the author has brought together he is clear, concise, and stimulating. In short, the work under review is one of the most important contributions to the subject made in recent times, and cannot fail to be of the greatest service to all who are interested in the normal and abnormal physiology of the nervous system.

In the opening chapter the phylogenetic development of the nervous system is considered, from its earliest appearance in medusæ upwards to the higher vertebrates, and the gradual growth of its importance in the individual economy. Next, the neuron theory is discussed at great length, the reasons for and against its acceptance being fairly stated with the views of the chief advocates on both sides, the author himself summing up, as a whole, in favour of the theory. Chapter iii. deals with reflex action generally, and with the spinal cord and the brain as reflex centres. Following this, the cord is considered as a central organ, and the significance of its segmentation on peripheral nerve distribution is pointed out, prominence being given to the more recent work of

Sherrington. The conducting tracts, afferent and efferent, are described in great detail as traced by the most recent anatomical and physiological methods, and the functions which each subserves, so far as they are known. Much space is given to the question of the crossing of the sensory tracts and of sensory conduction generally, the most important contributions to the subject from the time of Schiff till the appearance of Head's work being critically examined, but in the light of so much conflicting evidence it is difficult to make a final statement.

The rest of the book is taken up with the brain itself, the reflex centres of the medulla oblongata and the cerebellum and its connections receiving very full treatment. Four chapters are devoted to the cerebrum, dealing with motor and sensory cortical representation, and numerous cases illustrative of different forms of motor and sensory disturbance are given. In one chapter the primary and secondary motor paths are dealt with, and in another the different forms of speech disturbance, and the cortical lesions producing these. In the last chapter an account is given of the composition, formation, and uses of the cerebro-spinal fluid, and of conditions which may result from abnormal variations in its pressure.

The paper and printing are excellent, and the book is illustrated by one plate and eighty-one figures and diagrams, many of which are original. At the end there is an extensive bibliography for each chapter. Dealing largely as it does with abnormal phenomena, it ought to be in the hands of all physicians who make a special study of nervous and mental disease.

SUTHERLAND SIMPSON.

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Review
of
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Original Article

UNUSUAL SEQUELA OF HERPES ZOSTER (? POSTERIOR POLIOMYELITIS).

By ALEXANDER BRUCE, M.D., Etc.

In view of the attention which has been recently drawn, more especially by Déjerine, Thomas and Laminière, to the lesions of the spinal cord subsequent to herpes zoster, and as a contribution to the symptomatology of presumptive lesions of the posterior cornu, the following unusual case appears to be worthy of being recorded:—

On the 2nd of May of the present year, Miss C., stationer, aged 54, began to suffer from herpes zoster on the right side at the level of the 7th and 8th intercostal spaces. The attack was a very severe one, greatly prostrated the patient, and compelled her for three weeks to remain in bed. When she was allowed to get up she could walk quite well at first, but after a few days she observed a degree of weakness and a numb feeling in the right leg. This numbness was first felt below the knee, and then gradually passed up the right thigh and the right half of the trunk as far as the lower limit of the herpetic eruption. It then affected the corresponding part of the left side of the trunk, and extended downwards along the left lower extremity as far as the foot. Thereafter it began to diminish, and in a fortnight had almost entirely disappeared, only a slight tingling and numbness of the toes of the right foot remaining.

During this fortnight the right foot felt constantly cold, but when put to the ground gave a sensation as if there were hot pipes under the floor. The lower part of the back also felt very warm in bed, as if the patient were lying on hot bottles. She also noted that the lower part of the trunk and lower extremities had ceased to perspire.

Two or three days after the beginning of the numbness she drew the attention of her physician, Dr Thin, to her condition. On his first examination, Dr Thin discovered a degree of tactile anæsthesia over the right gluteal region and the upper part of the thigh. On a second examination made on June 12th by Dr Thin and myself, it was found that there was analgesia over the whole of the right lower extremity and of the right half of the trunk below the level of the herpes. It was stated also by the patient that her sensibility to temperature was defective—that when she put her foot into very hot water it felt as if it were cold. A careful examination showed that this thermoanæsthesia was present on the right side over the lower part of the abdomen and the right lower extremity, *i.e.* over practically the same area as that which was analgesic. Tactile sensibility, except over the area on the buttock and upper part of thigh already referred to, was perfect on the right side. The patient could feel the ground under her foot perfectly well. She was perfectly conscious of the position of her limbs, and could imitate with the left limb accurately any position into which its fellow on the right might be placed. There seemed to be no material diminution of muscular power while the patient was in bed, but when the attempt to walk was made there was a degree of weakness of the right lower limb.

The deep reflexes were exaggerated on the right side, and the plantar reflexes were extensor on the right side and flexor on the left. There was no affection of the sphincters.

As the patient was regarded as suffering from an unusual form of organic affection of the spinal cord, and as she could not be adequately nursed in her own house, her removal to the Royal Infirmary was recommended. She was admitted to that Institution five days later, on the 17th of June, and the following notes with regard to her case were made, with the assistance of my Resident Physician, Dr Hugh Wilson :—

The *family history* appears to be unimportant. Father died

at sixty-three of some unknown cause; mother at seventy-three of cancer in the abdomen. One brother is alive and well; a second brother died of consumption.

Patient suffered from measles, scarlet fever, and smallpox as a child, and from dry pleurisy two years ago; otherwise she has enjoyed good health, has had a comfortable home, light work, and sufficient food. She is a particularly intelligent, sensible woman, quite free from any of the stigmata of hysteria, and, apart from a somewhat exhausted look, evidently the result of her recent illness, she appears fairly healthy. There is no cyanosis or œdema anywhere.

The cicatrices of the herpes form a zone about 4 inches broad round the right side of the trunk from the middle line in front to the middle line posteriorly over the 7th and 8th intercostal spaces.

The circulatory system showed no abnormality, except an accentuation of the second sound of the heart in the pulmonary area. The pulse-rate was 80 per minute. The pulse was regular in time and force.

The respiratory system was practically normal, with the exception of a possible slight rise of pitch in percussion of the right apex.

The alimentary system was normal.

The urine was acid, showed a specific gravity of 1018, and contained no abnormal constituents.

Nervous System.—Sensory Functions.—On the examination of the sensory system it was now noted that the dissociation symptoms observed on the 12th of June were no longer limited to the right lower half of the trunk and right lower extremity, but had affected the corresponding parts of the left side in an identical manner. Careful testing elicited no loss of tactile or muscular sense on either side below a line slightly above the level of the umbilicus, with the exception of a small area over the right gluteal region and right upper part of the thigh. Here light touches were with some difficulty recognised. Elsewhere sensibility to touch was acute, and its localisation accurate. The muscular sense of both lower extremities was in no way impaired. Below the level of the umbilicus there was complete loss of sensibility to pain and to temperature, as indicated in the accompanying Figures 1 and 2. Between the level of the

umbilicus and that of the lower border of the herpetic eruption the loss of pain and thermal sense was not quite absolute.

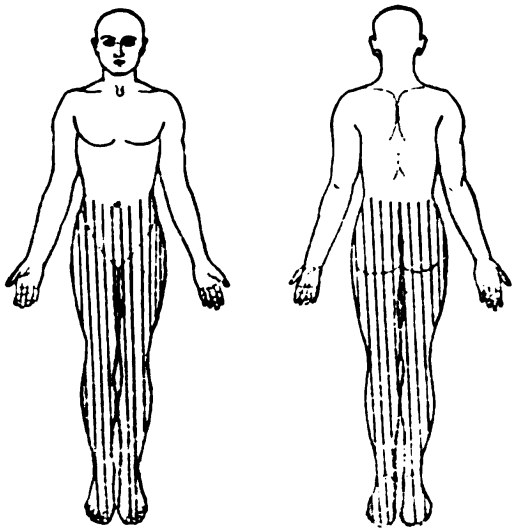

FIG. 1.—Vertical shading shows the area of analgesia on 12th June.

There was a complete absence of perspiration over the lower part of the trunk and the lower extremities. The patient said that when in her usual health she did not perspire readily, but her skin was now unusually dry all over this area.

The abdominal and epigastric reflexes were not elicited on either side. The plantar reflex was extensor in character, with separation of the smaller toes (*signe d'éventail* of Babinski) on both sides. There was a marked increase of the knee-jerks, Achilles-jerks, and adductor-jerks on both sides. Direct muscular irritability was also considerably increased. There was no ankle clonus.

Apart from the feeling of pain on the right side, due to the herpes, there was no girdle pain or feeling of constriction round the body.

Motor Functions. — While the patient lay in bed the strength of the lower limbs seemed to be fairly equal on both sides. It seemed somewhat impaired as tested by the power of pushing and drawing up the limb and by the movements at the ankles. The patient could not stand or walk without support. With support she could walk a few steps, then became greatly exhausted, and had

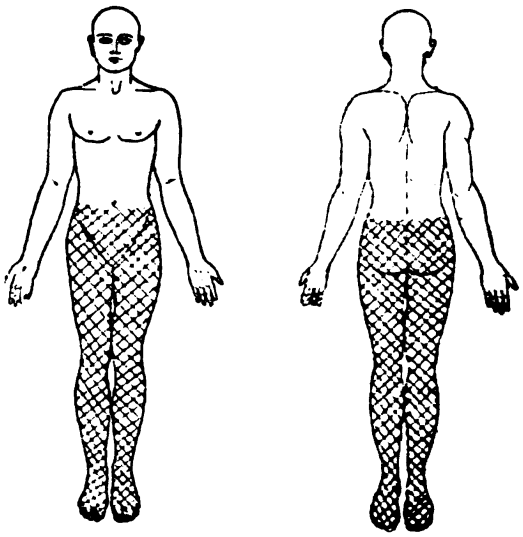

FIG. 2.—Area of anæsthesia to heat and cold on 12th June.

to be put back to bed. The muscles of the lower limbs felt somewhat flabby. There was no inco-ordination of the movements of the lower limbs. The circumference of each thigh was 13½ inches, that of each calf 10 inches.

There was no loss of power in the hands or arms. The biceps, triceps, and supinator jerks were equal on both sides, and were not exaggerated.

The functions of the cranial nerves were unaltered. There was no nystagmus. The pupils were equal and reacted to light and accommodation, and all the movements of the eye-ball were normal. The fundus oculi on both sides were normal. The field of vision was normal. There was slight pain on pressure above the left eye-brow.

The subsequent history of the patient during the time of her stay in hospital showed a somewhat rapid recovery from the motor symptoms, and a more gradual and less perfect subsidence of the sensory phenomena.

On the 19th June it was noted that she began to perceive a pin-prick on the dorsum of the right foot as painful, and on the same area she could recognise a stimulus of very hot water as such, but could not recognise cold as such, or detect a slight elevation of temperature. Above the dorsum of the foot her sensory symptoms remained unaltered.

The general condition had considerably improved, but not sufficiently to permit of her being allowed out of bed.

In the evening, patient noted that for the first time since the beginning of her illness her feet began to perspire. The perspiration was copious, and made the feet feel wet and cold.

On the 20th of June the analgesia and thermoanæsthesia were lost as regards the dorsum of the foot, and slightly diminished between the ankle and the knee of the right leg. Here the prick of a pin, or a pinch, or the application of a test-tube containing very hot water caused a slight tingling sensation which could not be further differentiated.

On being allowed to get out of bed, patient could walk a few steps with support, but felt very insecure and became readily exhausted.

On the following day painful and thermal stimuli were readily recognised as such over the lower part of the right leg, and slightly over a corresponding area in the left leg.

On the 23rd of June an area extending forwards from the spinal column posteriorly for about four inches along the 9th, 10th, and 11th ribs on the left side was noted to be red and œdematous, slightly raised above the surface, and very tender to pressure. The colour over the area was fairly uniform, was not accompanied by vesiculation, and partly disappeared on pressure.

Pain.—A pin-prick was felt sharply below the knee on the right side, and faintly over the right thigh as high as Poupart's ligament. Over the lower abdomen, between the level of the umbilicus and Poupart's ligament, pain was not perceived on either side, and there was complete analgesia on the left lower extremity as far down as a level slightly below the knee. There was a narrow zone of hyperalgesia above the level of the umbilicus.

Temperature.—Below both knees, but especially on the right side, differences of temperature could be recognised fairly well. On the thighs, and on the lower part of the abdomen to a level of about one inch higher than the upper limit of the analgesic area, heat was not recognised from cold. This level corresponded to a line drawn between the anterior extremities of the 9th ribs. Above this line a hot test-tube applied over the zone of the herpes caused both pain and a sense of heat.

Tactile Sensibility was quite normal, the area of partial anæsthesia on the right side having now disappeared.

Vaso-motor Symptoms.—A slightly heated test-tube caused ready and persistent flushing of the skin over the lower part of the abdomen on both sides.

Motor Functions.—Patient could now walk without support, taking short steps, evidently from dread of falling. There was no ataxia, and the muscular sense was quite normal.

During the following month the motor power in the lower limbs gradually improved, and does not call for special remark until about the beginning of August, when it was noted that the plantar reflexes had become definitely flexor in character. From this date up to the present time they have remained so. At no subsequent examination has any extensor response ever been elicited. The deep reflexes remained active, though not to the same degree as at first.

As regards the sensory functions, the tactile and muscular senses have remained throughout, and are at present (23rd November) absolutely normal.

The further history of the analgesic and thermo-anæsthetic areas is as follows :—

The analgesic zone gradually contracted from below upwards. On the 3rd of July it was limited to an area from about one inch above the umbilicus to the level of the lower part of the abdomen. This area of analgesia remained more or less constant until she left hospital on the 5th of September. By the 23rd of November the area of complete analgesia had entirely disappeared.

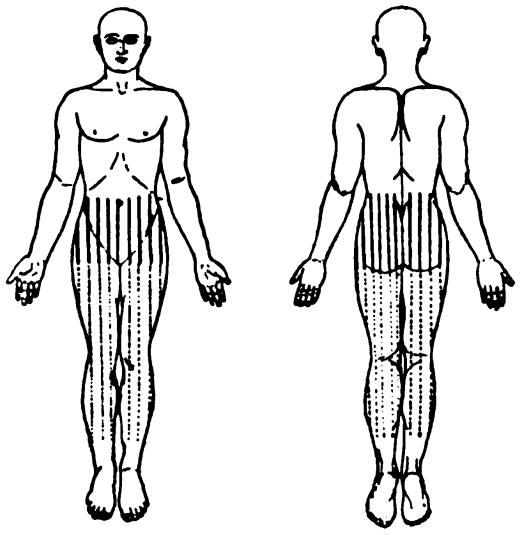

FIG. 3.—Vertical lines show area of analgesia found on 31st August. Vertical dotted lines show area of blunted pain sensibility.

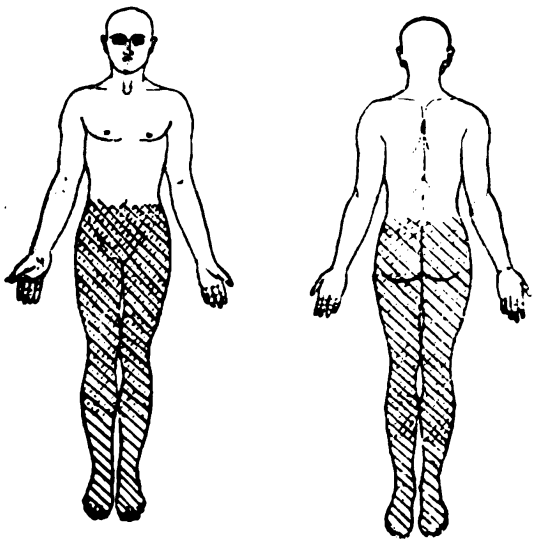

FIG. 4.—Oblique lines show area over which cold test-tube caused great hyperæsthesia. Oblique dotted lines correspond to the area of reduced and delayed sensibility to heat.

During July and August the thigh and upper part of the leg showed somewhat varying degrees of sensibility to pain. Occasionally when the patient was tired during the examination the sensibility was found to be again lost, but the condition most frequently present was a diminution of the acuteness of recognising a pin-prick as painful. That condition is indicated in Fig. 3 (dotted vertical lines).

At present (23rd November) the analgesia has entirely gone, but the prick of a pin is not so acutely felt over

the thighs and the lower part of the abdomen as elsewhere. As the analgesia diminished it was noted that stroking with a brush caused intense hyperæsthesia. This phenomenon gradually disappeared.

Temperature.—The perception of differences of temperature was somewhat irregularly regained. The area of thermoanæsthesia contracted from below upwards. Cold was recognised as such before heat. By the 9th of August the zone of complete thermoanæsthesia was limited to an area extending from the 9th rib to the level of the crest of the ilium. Below this a very hot test-tube caused pain and prickling as well as a sensation of heat, which was only recognised after some delay (Fig. 4).

On the 23rd of August it was noted that application of a cold test-tube applied to any part of the area which was primarily thermoanæsthetic caused a painful prickling sensation. On several occasions it was noted that there was an exaggerated sensibility to heat, cold, and to pin-pricks above the level of the herpes, but these exaggerations were very fugitive, and have now absolutely disappeared.

Status præsens.—Patient has entirely recovered her power of walking. The epigastric and abdominal reflexes have returned. The plantar reflexes are both flexor. The knee-jerks are somewhat active; the Achilles-jerk is present and active. There is no ankle clonus. There is no area of complete analgesia, although there is some diminution of the acuteness of pain-perception over the thighs and the lower part of the abdomen. There is slight confusion between the hot and cold test-tubes, but after a slight delay they can generally be accurately perceived except on the back of the left foot, where the cold tube is called hot and produces a painful stimulus. A very warm test-tube is well recognised as such, and also produces a painful cutting sensation.

Patient suffers greatly from troublesome paræsthesiæ; the thighs constantly feel as if burning, and the feet and lower parts of the legs as if encased in ice.

The general health is now very good.

Commentary.

When the patient was examined on the 12th of June by Dr Thin and myself the following symptoms were present and had to be accounted for:—

(1) Muscular weakness of the right lower limb;
(2) Exaggeration of the deep reflexes;
(3) Extensor plantar reflex;
(4) Loss of pain sense in the right lower limb and lower part of the right side of the trunk;
(5) Loss of thermal sense in the same region;
(6) Conservation of muscular sense; and
(7) Conservation of tactile sense in the same region (with the exception of a slight area of anæsthesia over the buttock and thigh).

The motor symptoms were of such a nature as to postulate the existence of an organic lesion as distinct from a mere functional disturbance, and to point to a lesion, moreover, involving some part of the upper motor neurones.

The sensory phenomena were practically identical with the dissociation symptoms so often found in syringomyelia, and pointed to a lesion of the base of the posterior horn.

The fact that the motor and sensory phenomena were on the same side of the cord and not on opposite sides, as in the Brown-Séquard syndrome, indicated that the path for the conduction of thermal and painful stimuli was involved before it had crossed to the opposite side of the cord, and further that the path from the opposite side was not involved.

The lesions in syringomyelia indicate that the loss of the conduction of painful and thermal stimuli with the conservation of the tactile and muscular sense depends upon a lesion at the base or in the substance of the posterior horn. A lesion, therefore, occurring in that situation would produce the sensory symptoms above noted.

The difficulty now arose of finding a point at which the pyramidal tract could be simultaneously involved. It is usually supposed that the pyramidal tract terminates by sending its fibres directly forwards and inwards towards the cells in the anterior cornua, but recent researches, in particular by Dr

Sutherland Simpson and Dr Jolly (1) in Professor Schäfer's laboratory in the University of Edinburgh, demonstrate that, in the monkey at any rate, this is not the case, but that in the cervical, thoracic, and lumbar regions the fibres pass from the pyramidal tract directly inwards to the outer half of the base of the posterior cornua. The accompanying illustration (Fig. 5), published with the consent of the authors of the paper and the Council of the Royal Society, Edinburgh, demonstrates this fact very clearly. It will be obvious that a lesion involving the base of the posterior cornua in the area containing the degenerated fibres would produce both the sensory and the motor symptoms above mentioned. It will be noted also that such a lesion would almost certainly involve part at least of the intermedio-lateral tract, and be responsible for the interference with the sweat secretion present at the beginning of the illness.

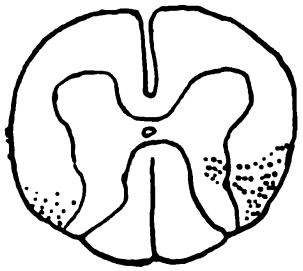

FIG. 5.—Degenerated fibres of the pyramidal tract extending into the base of the posterior cornua. (Sutherland Simpson and Jolly.)

The lesion, from its gradual onset and limitation, was probably of vascular origin, either thrombotic or inflammatory, arising in connection either with the postero-lateral or the posterior cornual arteries, or perhaps the mid-cornual branch of the central artery. Such a lesion causing a pressure upon and disturbance of function of the terminal fibres of the pyramidal tract, the cells involved in thermal and painful conduction, and those of the intermedio-lateral tract associated with the function of sweating and of vaso-motor control, would explain the symptoms completely (Fig. 6).

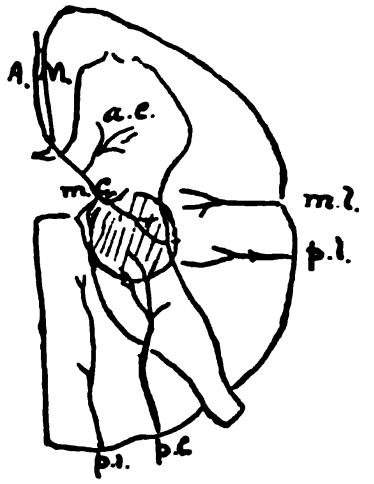

FIG. 6.—Diagram to show the vascular supply of the base of the posterior horn. (Sutherland Simpson and Jolly.) Circular area indicates site where a lesion might affect simultaneously the pyramidal tract and the centres for pain and temperature senses.

The subsequent extension to the other side could only be attributed to the occurrence of a similar lesion on the opposite side of the cord.

So much attention has been paid to the symptomatology of the vascular lesions of the anterior cornua that the explanation of affections of the posterior and lateral cornua have been largely overlooked. In a communication by Dr Harvey Pirie and myself (2) in the *Review of Neurology and Psychiatry* at the beginning of this year, attention was drawn to the association of the intermedio-lateral tract in the dorso-lumbar region with the function of sweating in the lower extremities. Since the patient was first seen and the diagnosis made, an interesting paper has been published by André-Thomas and Laminière (3), in which vascular congestion, hæmorrhagic foci, and disturbance of the grey matter on the side of a herpetic eruption and in the region of the lateral horn are described and figured. I have taken the liberty of reproducing one of their figures (Fig. 7), which shows the occurrence and site of such a hæmorrhage, and it will at once be seen that were such a hæmorrhage to extend downwards into the lower dorsal and lumbar region it would account exactly for the symptoms of the disease.

FIG. 7.—(Borrowed from André-Thomas and Laminière.) Shows hæmorrhage at the base of the posterior horn extending into the lateral horn.

The varying degrees of recovery of each function would depend upon the greater or less destructive action on the part of the original lesion.

Whether or not the view taken as regards the nature of the lesion be correct, the case is of interest as showing that herpes zoster is a condition which may leave effects more extensive than those due to the spinal ganglion in which the lesion originally occurred; and it will probably be found, on further investigation, that pathological changes are confined not merely to the posterior root-ganglion and to degenerations in the peripheral and sensory nerves consecutive to its destruction, as has been so well shown by Déjerine and André-Thomas (4), but that there are not infrequently widespread lesions within the cord itself.

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3. André-Thomas et Laminière. "Les Lésions Medullaires du Zona." *Rev. Neurol.*, July 30, 1907, p. 693.

4. Déjerine et André-Thomas. "Les Lesions radiculo-ganglionaires du Zona." *Rev. Neurol.*, May 30, 1907, p. 627.

Abstracts

ANATOMY.

THE PHYLOGENETIC DISPLACEMENT OF THE MOTOR
(474) **BULBAR NUCLEI; ITS CAUSE AND SIGNIFICANCE. (Phylogenetische Verlagerung der motorischen Oblongatakerne, ihre Ursache und Bedeutung.)** C. U. A. KAPPERS, *Neurolog. Centralbl.*, Sept. 16, 1907, S. 834.

WHILE the position of the somato- and viscero-sensory portions of the medulla remains fairly constant in all vertebrates, that of the motor nuclei is very variable. For instance the third, sixth, and twelfth nuclei lie in the ventral part of the medulla in fishes and reptiles, but in the higher vertebrates they have moved dorsalwards to the floor of the fourth ventricle. On the other hand the motor seventh nucleus and the nucleus ambiguus, which originally belonged to the viscero-motor system, lay in the dorsal portion of the medulla in the lower vertebrates, and have, during the course of phylogenesis, moved ventralwards and lateralwards; the intra-medullary course of their axones represents the direction in which they have been displaced.

This change of position may be most naturally explained by the functional connections of the nuclei. The ventralward displacement of the facial nucleus and of the nucleus ambiguus—which supplies fibres to the laryngeal and pharyngeal muscles—is due to the development of an efferent cortical system, these nuclei being intimately connected with it move towards it. The oculo-motor nuclei, on the other hand, being less dependent on cortical connections, have moved dorsalwards to come into closer connection with the association systems of the dorsal longitudinal bundles.

The dorsalward displacement of the twelfth nucleus is less easy to explain, but the author assumes that as taste is one of the chief functions of the tongue, the majority of its movements are reflex, and the nucleus has consequently moved nearer to the afferent centres of the sensory seventh (the nervus intermedius of mammals) and of the glosso-pharyngeal nerves.

GORDON HOLMES.

CONTRIBUTION TO THE STUDY OF THE HYPOPHYSIS
(475) H. JORIS, *L'Acad. roy. de méd. de Belgique*, Tome xix., f. 6.

THE author has examined this gland from the dog, cat, calf, guinea-pig, rabbit, sheep, and man, and the chief conclusions at which he arrives are: That the nervous lobe is a decayed structure composed of connective tissue and neuroglia, where, in contradistinction to Berkley's results, neither nerve cells or fibres are met with. The paranervous lobe is composed of emigrated ependymal cells. The fibrous and vascular structure is common to it and the preceding region, and the two together form the posterior lobe.

The anterior lobe is a true gland, and the great variety in the forms of its cells corresponds to different but coexisting stages of a single cell activity.

The vesicles do not represent a normal stage of a secreting process; they are due to a degeneration or involution of certain cells.

The pituitary stalk represents the vestiges of a part of the hypophysis existing in certain inferior vertebrates participating in the secretion of the cerebro-spinal fluid. Neither from embryological, histological, or cytological appearances is one justified in drawing a structural parallelism between the hypophysis and the thyroid gland.

JOHN TURNER.

PHYSIOLOGY.

BLOOD-PRESSURE AND THE NERVOUS SYSTEM. LEONARD
(476) WILLIAMS, *Clinical Journal*, Oct. 2, 1907.

THE physiology of the nervous control of the calibre of the blood-vessels is first briefly considered, *i.e.* the responsibility of the centres in the cord for the local variations which take place, as for example in digestion; and the limitation of the power of the medulla to the ordering of a generalised vaso-constriction or vaso-

dilation. The predominance of the interests of the medulla in the matter of blood-pressure is insisted upon, and instances are given of the action of the bulb in bringing about an increased arterial pressure in order to ensure a continuance of its own supplies. One such example is afforded by mitral stenosis, in which arterial pressure is invariably high in order that it may surmount the augmented venous pressure which obtains in that disorder. Another example is cerebral hæmorrhage, in which the arterial pressure is raised in order to overcome the resistance offered by the heightened intracranial pressure. This augmented arterial tension is an act of self-preservation on the part of the medulla and must not therefore be interfered with. If it is, as by nitrite of amyl or venæsection, the arterial pressure falls below the intracranial pressure, the medulla fails to receive its supplies and the patient dies.

Subnormal blood-pressures are due to medullary exhaustion. The bulb is no longer able to order a vaso-constriction, hence, in acute cases, shock and collapse; in chronic cases, neurasthenia. Drugs such as strychnine, which produce vaso-constriction by appealing to the exhausted medulla, should therefore be avoided in such cases, and reliance placed upon such direct vaso-constrictors as ergot and adrenalin. Brief reference is also made to the blood-pressure aspects of hysteria and insomnia.

AUTHOR'S ABSTRACT.

PATHOLOGY.

A BRAIN WITH COMPLETE ABSENCE OF THE CORPUS CALLOSUM. (477) JOHN CAMERON, *Journ. of Anat. and Phys.*, Vol. xli., 1907, p. 293.

OF the thirty cases of deficiency of the corpus callosum published up to 1888, Bruce ("On the Absence of the Corpus Callosum in the Human Brain," *Proc. Roy. Soc. Edin.*, Vol. xv., 1888; *Brain*, Vol. xii., 1889) found that entire absence occurred only in fifteen of these. In the present case the corpus callosum was completely absent.

The main interest in this specimen is centred in the commissural structures which are developed in association with the lamina terminalis. Thus, in addition to the absence of the corpus callosum, there is deficiency of the transverse psalterial fibres of the fornix, the two lateral halves of the latter being connected merely by an exceedingly thin semi-transparent membrane. The "fore and aft" fibres of the fornix are therefore alone represented. From the outer margin of the band of fibres representing each lateral half of the body of the fornix there is an extension outwards of the thin membranous sheet already referred to. This

can be readily traced to its termination by blending with the cortex of the callosal convolution at the bottom of what corresponds to the callosal sulcus. It accordingly represents the *lamina terminalis*, which has retained its embryonic attachments in this specimen.

Two other longitudinal or "fore and aft" systems of fibres run in each lateral half of the lamina terminalis close to the outer aspect of the fornix, and lying more or less parallel to the latter. The band next to the fornix represents the *striæ longitudinales*, while the attachments of the most externally placed system correspond to those of the *cingulum*.

The intimate association of the fornix, longitudinal striæ, and cingulum with the lamina terminalis suggests that in this case they had probably all been developed in that structure.

AUTHOR'S ABSTRACT.

(478) **CONCERNING THE BEHAVIOUR OF THE MOST PROXIMAL PART (EXTRA-MEDULLARY AND EXTRA-PIAL) OF THE POSTERIOR ROOT TOWARDS DEGENERATION AND REGENERATION.** [**Ueber das Verhalten des proximalsten (extramedullären und -pialen) Teiles der hinteren Wurzeln bei Degeneration und Regeneration.**] G. BIKELES, *Neurol. Centralbl.*, No. 20, 1907.

BIKELES found in a dog, which survived for 36 days after compression of the posterior root at the proximal end of the spinal ganglia, marked degeneration over a considerable length of the root, which remained unstained by Weigert, and showed scarcely any trace of products of degenerated myeline; but a small segment lying immediately behind the place of entry of the root through the pia stained tolerably well, and just like the intra-medullary continuation of the root. The condition was exactly reversed in two animals kept alive for three months after compression of their posterior roots. These showed at the place of compression very numerous, and lying tolerably close together, fine regenerated nerve fibres, which could be traced through a considerable length of the extra-medullary root, up to not far from its point of entry through the pia; but at a slight distance from the pia (and therefore extra-pial) the number of regenerated nerve fibres diminished very much, with at the same time an increase of products of degenerated myeline, so that this part of the root was not distinguishable in appearance from that of the intra-medullary part. This extra-medullary segment of the root, which shows on the one hand a slow course of degeneration, and on the other

only scanty regeneration, is clearly marked off from the rest of the extra-medullary root, and the boundary between these two parts is in sections stained by Weigert-Pal a quite sharp line generally convex on its peripheral side.

Bikeles thinks that here is evidently a similar condition to that which has recently been described by E. Levi in man; that is to say, a continuation of the glia, together with an absence of Schwann's sheaths on the still extra-pial lumbar hind root, so that the boundary between the glia and the end of the Schwann's sheaths corresponds with the dividing line of Obersteiner and Redlich.

The absence of the sheath of Schwann on the most proximal segment of the extra-medullary lumbar posterior roots may be the chief cause why in reference to degeneration and regeneration this part no longer behaves like a peripheral but like a central nerve.

(Levi particularly remarks on the involvement of this segment in tabes dorsalis.) JOHN TURNER.

NEUROPHAGY IN GRAFTS OF SPINAL GANGLIONS. (**Neurophagie dans les greffes de ganglions rachidiens.**) J. NAGEOTTE, *Rev. Neurol.*, Sept. 15, 1907, p. 933.

(479)

NAGEOTTE concludes from his experiments with grafts of spinal ganglia that the star cells of Cajal, and not the endothelial cells, are the special phagocytes of the dead nerve cells. These together constitute the satellite cells. He is not certain whether the Cajal cells are ectodermal (neuroglial) in origin, or mesodermal; but it is certain if they are mesodermal that their immigration is anterior to all morbid phenomena, since they are normal inhabitants of the subcapsular spaces. At the first appearance of phagocytosis they spread out their branches over the surface of the nerve cell, and push forward a prolongation which perforates it and hollows out in it a cylindrical passage, then the nucleus is drawn into this prolongation, and finally the entire cell lies within the dead nerve cell. Eventually by the action of several Cajal cells the nerve cell is hollowed out into a network of anastomosing passages.

The inclusion of the nerve cell nucleus takes place by a cell of Cajal forming a crescent, of which the two ends bend round and reunite behind the nucleus and thus enclose it in a vacuole. The nucleus becomes angular, reduced in volume, and finally disappears. Other cells of Cajal within the nerve cell send out excessively fine protoplasmic filaments which, reuniting at their extremities, form a series of vacuoles, in which fragments of the nerve cell lie. In this way the entire nerve cell ends by being absorbed.

He finds a deposition of fatty granules in (1) the satellite cells of the dead nerve cells; (2) in the Cajal cells in the interior of worm-eaten nerve cells; (3) in the satellite cells of certain surviving nerve cells, in the vicinity of dead cells; (4) in a very few nerve cells, living or dead; (5) in the connective tissue cells of the surrounding capsule, in the vicinity of the fatty satellite cells.

This fatty deposition is either the expression of a peculiar pathological reaction of these cells which shows itself by the intra-cellular secretion of a fatty substance capable to a certain extent of diffusion, or is the result of the transformation of substances from the nerve cell at the moment of its death. He thinks that the latter supposition is the more probable.

Nageotte believes that the networks which he describes in the dead cells certainly correspond to a normal disposition and structure of the nerve cells. They present a striking analogy to Holmgren's "trophospongium" and to the endocellular network of Golgi.

With regard to the relation between this endocellular formation and the Cajal cells during the first phases of phagocytosis, three suppositions are permissible:—

(1) That the cells of Cajal enter these canals because of the facility offered by the existence of a gaping passage, which they have only to dilate.

(2) That the network contains a substance for which the cells of Cajal are particularly eager.

(3) That the network is formed as Holmgren supposes by protoplasmic prolongations of subcapsular cells, and the progress of perforating cells will then be merely a transport of the cell body along one of these prolongations. If this hypothesis is verified, one would be justified in admitting that the trophospongium of Holmgren is constituted by the cells of Cajal and not by the endothelial cells. JOHN TURNER.

CLINICAL NEUROLOGY.

ACUTE SECONDARY SYPHILITIC MENINGITIS. (Méningite syphilitique secondaire aiguë.) (480) BOIDIN et WEILL, *Presse Médicale*, Oct. 19, 1907, p. 681.

A MALE, aged 18, was admitted to hospital with all the symptoms of acute meningitis. The case at first appeared to be one of tuberculous meningitis. Examination, however, revealed a hard chancre on the penis, but no other signs of syphilis were present.

Lumbar puncture showed almost pure lymphocytosis. Four days later the roseola appeared. The diagnosis of syphilitic meningitis was confirmed by examination of the patient's wife, who, in addition to the scar of a vulvar chancre, presented enlarged occipital glands, mucous tubercles in the mouth, and an intense roseola. Under the influence of daily injections of biniodide of mercury rapid recovery took place. The chronology of the disease was as follows: Chancre, middle of June; headache, July 15; meningeal symptoms, August 5; roseola, August 12; recovery from meningitis, August 17. As on previous occasions (vide *Rev. of Neurol.*, September 1907, p. 718), examination of the cerebro-spinal fluid for the spirochœta pallida proved negative. The pathogeny of the meningeal affection in the secondary period of syphilis can be explained by the fact that the syphilitic virus has a predilection for the ectoderm, from which both the skin and the nervous system are derived. The meningeal irritation may be regarded as a true exanthem which is often latent, but which may manifest itself apart from lymphocytosis by more or less numerous and acute symptoms. J. D. ROLLESTON.

THE SYMPTOMATOLOGY OF LESIONS OF THE LENTICULAR
(481) **ZONE, WITH SOME DISCUSSION OF THE PATHOLOGY OF APHASIA.** CHAS. K. MILLS and WM. G. SPILLER, *Journ. Nerv. and Ment. Dis.*, Oct. 1907, p. 624.

THE attempt is made in this paper to reach some definite conclusions concerning the functions of the lenticular nucleus. The paper is based upon the study of eleven cases with necropsy, in ten of which the lesion involved the lenticular zone. In six of these cases the lenticula itself was involved. Other parts of the lenticular zone were variously implicated in the ten cases. It was possible, at least in some of the cases, in which regions other than the lenticula were affected, to arrive at an idea as to the part played by this body in the symptomatology presented by the patients.

The views of many investigators concerning the functions of the lenticula are given, and the attempt is made to show that the island of Reil and the striatum are distinct anatomically and functionally; therefore they should not be considered together in discussing disturbances of speech.

The conclusions to which the authors come, from a study of their cases and of the literature of the subject, are as follows:—

1. Lesions restricted to the lenticula apparently do not cause sensory symptoms.

2. Motor symptoms probably result from lesions situated in certain parts of the lenticula; speaking generally, the lenticula may be regarded as a motor organ.

3. Anarthric or dysarthric speech disorders result from lesions of some portion of the left lenticula, which probably contains centres which are concerned with the movements which make speech possible.

4. Destructive lesions of certain portions of the lenticula probably cause a paresis of the limbs or face.

5. The paresis or paralysis caused by destructive lesions of the lenticula differs from that produced by capsular lesions, the impairment of power not being so severe and not being so characteristic in the former as in the latter case.

6. The paresis or paralysis which is caused by lenticular lesions differs from that produced by cortical lesions in that it is less likely to be dissociated; although dissociated lenticular paresis may occur.

7. While the loss of power which results from a destructive lenticular lesion is permanent, it is usually not intense.

8. Persistent true motor aphasia, as this form of speech disorder is generally understood, is not caused by a lesion restricted to the lenticula, no matter what its size or destructiveness.

9. The insula, cortex, and subcortex play an important part in speech phenomena, one entirely different from that played by the lenticula and the internal capsule.

10. The insula is a part of the cortical motor centre for speech, Broca's convolution probably forming with the insula the entire cortical motor centre for speech.

11. Motor aphasia may be present without a lesion of the left third frontal convolution.

12. The lenticula forms too large a portion of the cerebral hemisphere to be regarded merely as a vestigial organ.

AUTHOR'S ABSTRACT.

INTERMITTENT CLAUDICATION OF CEREBRAL ORIGIN.
(482) MEEUS, *Rev. Neurol.*, Sept. 30, 1907, p. 988.

A FORTY-TWO years old general paralytic showed, two or three times a day, temporary paralysis of the right side of the body, with paresis of the tongue and dysphagia. The paralysis rarely lasted longer than a few minutes, and the patient resumed his occupation as though nothing had happened.

An intermittent local ischæmia of the cortex is suggested.

S. A. K. WILSON.

THE CEREBELLAR-VESTIBULAR SYNDROME. CORIAT, *Am.* (483) *Journ. of Insan.*, Jan. 1907, p. 319.

BY the term "central ataxia" is meant an ataxia combined with disorders of equilibrium and locomotion, and an inability to orient the body in space. The chief symptoms would be agonising vertigo or sense of rotation, nausea, inability correctly to appreciate rotation in a horizontal plane with the eyes closed, and a cerebellar-ataxic gait. Included in this category are the labyrinthine ataxia of Dejerine, the vestibular ataxia of Raymond and Egger, Menière's disease, sea-sickness, etc.

The author describes a case of chronic suppurative otitis media with perforation of the membrana tympani, in which symptoms of grave cerebral disturbance more or less suddenly supervened. Unfortunately the patient's mental condition was apparently so unsatisfactory, that the value of the case, from the point of view of the ostensible subject of the paper, is considerably diminished. Moreover, a post-mortem examination revealed a "large subdural hæmorrhage over the right hemisphere, but normal middle and internal ears," and "serial sections of the cerebrum, pons, mid-brain, and cerebellum showed absolutely no lesions."

One is at a loss to understand why such a case should be recorded to illustrate the cerebellar-vestibular syndrome. The author attributes the symptoms to "pressure."

S. A. K. WILSON.

ON DISEASES OF THE LABYRINTH AS CAUSE OF SPASMODIC (484) **TORTICOLLIS. (Ueber Labyrintherkrankungen als Ursache der spastischen Torticollis.)** H. CURSCHMANN, *Deutsche Zeitsch. f. Nervenh.*, 1907, H. 3-4, p. 305.

OUR knowledge of the real etiology of spasmodic torticollis is still very deficient. Treatment of the affection, as a rule, is unsatisfactory, and the prognosis is correspondingly unfavourable. To regard torticollis as a product of hysteria is unjustifiable, for rarely do we succeed in curing the disease by suggestive therapeutics, even when pushed to the stage of hypnosis. Curschmann is therefore of opinion that underlying every case there must be an organic basis of some sort. Such organic cause may be found to exist in the labyrinth, as in three cases here recorded.

The first case was that of a hotel-keeper, 37 years of age, who from the age of 15 had suffered from gradually progressive right-sided deafness, following an acute inflammation of the middle

and inner ear. Together with the deafness he had giddiness and occasional attacks of Menière's syndrome. The permanent vertigo was most severe when the patient tried to lean his head towards the right; it was least troublesome when he leaned his head to the left, whereby the left sternomastoid turned the chin to the right, and the left ear was depressed towards the shoulder. Curschmann explains this phenomenon as the result of changes, probably of hydrostatic nature, produced in the diseased semicircular canal, whereby labyrinthine vertigo was produced on leaning the head towards the right. In this way there developed a true spasmodic torticollis, with hypertrophy of the over-acting muscles. Quinine in doses of five grains three times a day was administered, in view of its well-known palliative effect upon labyrinthine irritation. As a result, within a few weeks the giddiness and torticollis gradually disappeared. The patient had a relapse one and a half years later, which again yielded to quinine, although incompletely on this occasion.

The second and third cases were less severe. Both presented a similar clinical picture—undoubted labyrinthine phenomena of minor severity (chiefly in the form of irritative symptoms), horizontal vertigo, tinnitus, etc.—without gross auditory defects in the way of deafness. In the second case there was typical labyrinthine nystagmus towards the side of the more intense subjective symptoms. In both cases vertigo preceded the appearance of torticollis. The tendency to torticollis was directly proportional to the intensity of the vertigo. In both of these cases, as in the first, quinine had a beneficial effect, firstly on the vertigo, and afterwards on the tendency to torticollis. PURVES STEWART.

A CASE OF FACIAL HEMIATROPHY WITH OCULAR SYMP-
(485) **TOMS. (Ein Fall von Hemiatrophia facialis progressiva mit Augennervensymptomen.)** S. SALOMON, *Neurolog. Centralbl.*, Sept. 16, 1907, S. 846.

A GIRL, nine years of age, came under observation with the history of increasing asymmetry of the face for the past three years. There was considerable shrinkage of the whole left side of the face, but chiefly of the supra- and infra-maxillary regions. This was apparently due to the small size of the bones, as well as to atrophy of the muscles and disappearance of the subcutaneous fat. The skin of the left side of the face was also pale, thin, and atrophic, but there was no difference between sweat secretion or the amount of hair in the two sides. The electrical reactions of the smaller muscles of the left side were feeble. Sensation was intact on both sides of the face, and there was no evidence of

cervical sympathetic paralysis. The right external and the left internal rectus were weak, and the left pupil did not contract to light or on accommodation.

The author assumes that the presence of these ocular palsies indicates the neuritic origin of the facial hemiatrophy. It seems almost certain that the patient had congenital syphilis.

GORDON HOLMES.

HERPES IN DIPHTHERIA. J. D. ROLLESTON, *Brit. Journ. Derm.*, (486) Nov. 1907, p. 375.

IN 1370 consecutive cases of diphtheria, herpes facialis mainly affecting the lips occurred in 55 cases, or 4·01 per cent. It was almost invariably an early symptom coinciding with the angina. The usual date for its appearance was the third or fourth day. Herpes zoster was much rarer. It was noted in only two cases. In both it occurred on the twenty-ninth day, in each case after an angina of moderate severity. A striking parallel is afforded by lobar pneumonia, in which herpes labialis is so common while herpes zoster is very rare. Herpes labialis was found to be commoner in the severe than in the mild forms of diphtheria, though the phenomenon in itself in no way added to the severity of the disease. Neither were the lips invaded by membrane, nor did labial paralysis subsequently occur. Though paralysis of the lips is an occasional event in diphtheria—it was found in 37 of the 1370 cases—none of the cases with herpes labialis developed this complication. The pathogeny of herpes facialis and its relation to herpes zoster are still obscure. The greater frequency of herpes labialis in the severer forms of diphtheria is in favour of the hypothesis of Orsi, who regarded it as a cutaneous manifestation of a reflex originating in the pharyngeal or laryngeal mucosæ, the nerve terminals of which on receiving an abnormal stimulus generate vaso-motor disturbance in the skin resulting in the appearance of herpes. AUTHOR'S ABSTRACT.

HERPES ZOSTER AND PARALYSIS. *The Hospital*, October 26, (487) 1907, p. 85.

IN regard to this disease little has been written of any affection of the anterior nerve roots or motor nerve trunks simultaneously with the trouble in the posterior nerve roots. Motor trouble can and does occur in some cases. In cervical or trigeminal herpes facial paralysis or ophthalmoplegia may accompany or follow the erup-

tion. The prognosis of the paresis occasionally associated with herpes is very fair: the paresis may be transient, while in severer cases recovery begins some twelve or fourteen weeks after the paralysis sets in.

In one case of typical ophthalmic zona, the patient noticed on the tenth day, when the eruption was subsiding, that he could not get his left eyelid up to its full extent. During the third week from the commencement of the mischief he could not raise his left eyelid voluntarily at all. There was no paresis of any of the muscles moving the eyeball, nor any of the intraocular muscles. The trouble slowly diminished in intensity, and in about three months' time the ptosis had entirely disappeared, and the patient seemed absolutely well again in all respects.

In a second case, eight days after the appearance of a typical zona in the cervico-occipital region, it was noticed that the left side of the patient's face looked queer, and a day later there was complete infranuclear paralysis of the left facial nerve, except for preservation of taste. Seven weeks later the paralysis was still discernible, though much improved; ultimately it got perfectly well.

In a third case of zona, affecting the upper branches of the left superficial cervical plexus, there was intense irritation, and at the same time deficient sensibility in the region involved, and the hair fell out over it, but not elsewhere. A few days later it was noticed the patient could not close his left eye properly, and next day he had complete paralysis of the left facial nerve, with loss of taste on the left side of his tongue. No other cranial nerve was affected.

It seems not unlikely that the acute inflammatory exudation within as well as upon the skin in the last two cases affected much deeper structures also, and among them the periosteum at the stylo-mastoid foramen; this might be the explanation of the facial paralysis in the case where there was no loss of taste. To account for the loss of taste in the third case, one might assume that the inflammation extended a good deal further into the aqueductus Fallopii, reaching as far as the chorda tympani; in the first case perhaps the inflammatory exudate actually infiltrated the nerve going to the levator palpebræ superioris. S. A. K. WILSON.

(488) **UNILATERAL FACIAL HERPES AND CERVICAL SYMPATHETIC SYNDROME IN A CASE OF TABES.** JEANSELME SÉZARY, *Rev. Neurol.*, Oct. 15, 1907, p. 1029.

A case of tabes with a herpetic eruption on the left ear and on the left side of the lips. The left half of the face was red and shiny,

and the left ear was injected. There was myosis of the left eye. The temperature of the affected side was elevated.

Possibly the nerve filaments leaving the cord to reach the sympathetic by the eighth cervical and first dorsal were involved in the tabetic process. On the other hand, the Gasserian ganglion receives fibres from the cervical sympathetic.

S. A. K. WILSON.

OCULOMOTOR PHENOMENA OF CUTANEOUS LABYRINTHINE (489) AND COCHLEAR ORIGIN IN A CASE OF TABES.
ETIENNE, *Rev. Neurol.*, Oct. 15, 1907, p. 1025.

IN a characteristic and advanced case of tabes, there was diminution of auditory acuity on the left side, with tinnitus aurium on both sides. In addition, the patient had double optic atrophy, loss of pupil reflexes, divergent strabismus and diplopia. The divergent strabismus varied considerably in degree from time to time. Whenever the patient closed his eyes, dancing nystagmoid movements of the ocular globes at once took place, and continued some little time. Any passive displacement of the head produced similar oscillation of the eyes. Any fairly loud sound, such as the rumble of wheels in the street, inevitably caused the dance of the globes to reappear, and it continued while the sound lasted. Pinching the skin also served to elicit these sudden oscillatory movements of the eyes, which, be it remarked again, were noticed only when the eyelids were closed.

Careful and delicate examination by electrical methods made it clear that there was no muscular insufficiency of neuritic or myopathic origin as far as the oculomotor musculature was concerned.

Evidently it is the labyrinthine disease which explains the strabismus. From the nucleus of the vestibular nerve goes a group of fibres to the nucleus of the sixth on the same side. Hence the abnormal stimulation of the left sixth, revealing itself in contracture of the external rectus. Hence also tho determination of fresh ocular movements by modifications of the labyrinthine position.

These same cochlear or labyrinthine lesions explain the nystagmoid movements when the eyes are closed. If the attitude of the eyeballs is no longer fixed by active vision, they respond to other stimuli. The sixth nucleus, after receiving fibres from the vestibular nerve and from Deiter's nucleus, sends branches to the fourth and third on the same side. Hence ataxic movements may be aroused once the eyes are closed. By means of the posterior longitudinal fasciculus the opposite eye may also partake in the ataxia. Stimulation of the anterior nucleus of the cochlear nerve

may explain the ataxic movements of the globes in response to sounds.

As regards the nystagmoid movements consequent on stimulation of the skin (a phenomenon noticed by the author in two other cases which presented Romberg's sign), it is to be noted that the cells of the column of Clarke receive peripheral sensory stimuli and transmit them towards Deiter's nucleus and the cerebellum. The tabetic lesion in this case affects this system, with the result that the sets of fibres passing from Deiter's nucleus to the oculomotor nuclei convey defective peripheral sensory impressions. The normal reflex equilibrium of the nuclei is disturbed, and ataxia results.

S. A. K. WILSON

PRURITUS IN TABES. (**Le prurit tabétique.**) MILIAN, *Bull. et* (490) *Mém. de la Soc. Méd. des Hôp. de Paris*, Oct. 17, 1907, p. 991 LAMY, *Ibid.*, Oct. 24, p. 1015.

PRURITUS as a symptom of tabes is not mentioned either by neurologists or dermatologists, with the single exception of Jacquet.

Out of twenty-five cases of pruritus, independent of any skin lesion, Milian found tabes was the cause in six. Three of these were cases of pruritus ani, one was a case of pruritus ani associated with periorbital pruritus, the fifth was one of lumbar and thoracic pruritus, and in the sixth the epigastric region was affected. Tabetic pruritus is never generalised like the senile or toxic variety, but is always localised. It is a very obstinate symptom, lasting for years, and presenting paroxysmal exacerbations of extreme severity, especially in the case of pruritus ani. The pruritus may be a solitary symptom or be associated with a visceral paroxysm in the corresponding region, *e.g.* vesical tenesmus may accompany perineal pruritus, or gastric crises may coexist with epigastric pruritus. Other sensory disturbances may also be present in the region affected by pruritus, such as lightning pains and superficial or deep anæsthesia. In Milian's cases pruritus was always an early symptom, occurring only in those in whom the symptomatology was incomplete (tabétiques frustes). He never found it in those in whom ataxia had developed. Relief was obtained by exhibiting the drugs usually employed for the lightning pains, such as antipyrine, pyramidon, salicylate and nitrite of soda. Lumbar puncture proved still more effectual. Gastronomic and venereal indulgence were forbidden.

Lamy's case resembled those reported by Milian, in that the pruritus was localised and paroxysmal, but differed from them in that his patient had reached the last stage of tabetic cachexia and inco-ordination.

J. D. ROLLESTON.

THE CONDITION OF THE SUPERFICIAL AND DEEP REFLEXES

(491) **IN SOME ACUTE INFECTIONS. (Del modo di comportarsi dei riflessi superficiali e profundi in alcune infezioni acute.)** LIVIERATO, *Gazz. degli Osped.*, Aug. 25, 1907, p. 1062.

IN the course of acute infections alterations can easily be found in the condition of the superficial and deep reflexes. Such alterations are transitory, being most marked during the height of the disease and disappearing in convalescence, and are probably due to a purely functional lesion of the nervous system generated by the circulating toxines. The author investigated the knee-jerks and abdominal reflex in typhoid fever, lobar pneumonia, erysipelas, influenza, acute rheumatism, and measles. He selected young subjects free from organic nervous disease, excluding children and old persons, because in the former there is a marked diffusion of the reflexes, and in the latter the abdominal reflex tends to be ill-marked. Out of 46 cases of typhoid in which the diagnosis was based on clinical and bacteriological evidence, in 39 there was a diminution of the cutaneous reflexes and a contemporary exaggeration of the knee-jerks. In four cases the reverse occurred. In two cases there was a simultaneous exaggeration or diminution of both cutaneous and deep reflexes. Out of 42 cases of lobar pneumonia, in 23 an impairment of the activity of the tendon-jerks coexisted with a more or less marked exaggeration of the superficial reflexes. In 7 cases both the superficial and deep reflexes were more or less diminished. In the remainder exaggeration of the deep reflexes coexisted with diminution of the superficial. In erysipelas and in influenza the dissociation between the two categories of reflexes was not so marked as in typhoid and pneumonia. In 17 out of 22 cases of erysipelas the superficial reflexes were somewhat sluggish, while the deep were usually normal and rarely exaggerated. In 3 cases both were normal. In 2 cases exaggeration of the superficial reflexes was associated with diminution of the deep. In 40 out of 52 cases of influenza there was a more or less marked sluggishness of the deep reflexes. In 23 of these the superficial reflexes were normal, and in 17 they were exaggerated. In 9 cases both superficial and deep reflexes were sluggish. In only 3 cases exaggeration of the deep reflexes was associated with normal superficial reflexes. In 11 out of 19 cases of acute rheumatism exaggeration of the deep reflexes accompanied temporary diminution of the superficial reflexes. In 5 cases exaggeration of the deep reflexes was associated with a normal condition of the superficial. In the remainder there was either a contemporary exaggeration of the superficial and deep

reflexes, or an exaggeration of the superficial reflexes was associated with normal or diminished deep reflexes. In 5 out of 11 cases of measles exaggeration of the superficial reflexes was accompanied by diminution of the deep reflexes. In 4 cases the reverse occurred, and in 2 cases both reflexes were sluggish. It is noteworthy that Babinski's sign was negative in all the cases. There did not appear to be a direct relation between the gravity of the disease and the degree of dissociation of the reflexes, nor did the degree of dissociation seem to have any diagnostic or prognostic value. J. D. ROLLESTON.

(492) **ON THE EXAGGERATION OF CUTANEOUS REFLEXES ON THE PARETIC SIDE IN ORGANIC HEMI-PARESIS. (Ueber Steigerung der Hautreflexe auf der paretischen Seite bei organischen Hemiparesen.)** REDLICH, *Neurol. Centralbl.*, May 1, 1905, p. 395.

IN certain cases of organic hemi-paresis the writer found that the cutaneous reflexes were not, as is the rule, absent or diminished on the affected side, but were either decidedly more active than on the sound side, or that some of the reflexes were diminished, while others were increased. In five out of the seven illustrative cases Jacksonian epilepsy was present. The following hypothesis is put forward as an explanation. The reflex path for cutaneous reflexes passes through the cortex. In severe hemiplegia the reflexes are lost or diminished, because the cortex is destroyed or damaged. In hemi-paresis, on the other hand, an irritative condition only exists. The cutaneous reflexes are, therefore, exaggerated on the corresponding side, just as in irritative conditions of the spinal cord the tendon jerks are exaggerated on the affected side. This hypothesis the author admits does not explain how it is that the skin reflexes are not always exaggerated on the affected side in Jacksonian epilepsy, nor does it account for the occasional exaggeration of the skin reflexes on the paralysed side in severe hemiplegia. J. D. ROLLESTON.

THE PHYSIOLOGY OF THE THYROID GLAND IN ITS RELA-
(493) **TION TO EXOPHTHALMIC GOITRE.** S. P. BEEBE, *Journ. Am. Med. Ass.*, Oct. 5, 1907, p. 1155.

THE PATHOLOGY OF EXOPHTHALMIC GOITRE. W. G. MAC-
(494) CALLUM, *Journ. Am. Med. Ass.*, Oct. 5, 1907, p. 1158.

THE DIAGNOSIS OF EXOPHTHALMIC GOITRE. L. F. BARKER,
(495) *Journ. Am. Med. Ass.*, Oct. 12, 1907, p. 1235.

MEDICAL TREATMENT OF EXOPHTHALMIC GOITRE. R. B.
(496) PREBLE, *Journ. Am. Med. Ass.*, Oct. 12, 1907, p. 1238.

THE SURGICAL TREATMENT OF EXOPHTHALMIC GOITRE.
(497) ALBERT KOCHER, *Journ. Am. Med. Ass.*, Oct. 12, 1907, p. 1240.

THE general impression left on one after reading these papers, communicated at a symposium of the American Medical Association, is one of vast ignorance of the true inwardness of exophthalmic goitre. This is particularly the case with regard to the physiology of the thyroid; as Dr Beebe says, "we know very little of what the thyroid secretion does when confined within physiological limits, but if given in excess we see as a result many of those symptoms characteristic of exophthalmic goitre." He concludes that the hypersecretion theory offers the most satisfactory explanation of the symptoms in the disease, but that, as yet, we have no satisfactory explanation of the mechanism by which they are brought about, nor yet an explanation of the origin of the hyperactivity of the gland. There are two commonly accepted possibilities. First, as a result of nervous shock; second, as a compensatory hypertrophy during a toxæmia.

Dr MacCallum arrives at practically the same point by a different path. From an analysis of sixty cases he concludes that the only constant changes are those found in the thyroid, the thymus, and the lymphoid apparatus. With definite symptoms of exophthalmic goitre there is always the same change in the thyroid either in whole or in part; anatomically the change is the same as that found in compensatory hypertrophy (but without symptoms) after removal of a part of the gland. A mere hypertrophy, therefore, will not account for the symptomatology; we are almost forced to the belief that excessive activity is at fault.

But again, when we ask, "Whence the over-activity?" we are faced by a shut gate. There are no examples elsewhere of the spontaneous hypertrophy and over-activity of an organ to the detriment of the rest of the body. Always it is a compensatory hypertrophy, but here the inconsistency appears, that when part

of the hypertrophied gland is excised the symptoms often disappear. Experimental attempts to isolate the thyroid from all nervous connections, particularly sympathetic, have so far led to no result.

With Dr Barker on the diagnosis we stand on rather firmer ground. Of the cardinal signs, he lays greatest stress on the increased rate of the heart-beat; of the goitre which is *almost* always present to some extent the vascular peculiarities are more important from the diagnostic standpoint than the enlargement. Many of the phenomena can be brought into line with disturbances of the autonomic neurone systems. Of eye signs he notes no fewer than eleven, all or any of which may be present. His last paragraph deals with the diagnosis of the indications and contra-indications for surgical interference. Here he says: "In the present state of knowledge and practice, once a positive diagnosis has been made, it is the physician's duty and privilege to recommend operation early . . . but a heavy responsibility rests with the physician, for fully as important as the diagnosis of the indication for operation is the choice of the surgeon." The only contra-indications are a feeble heart and high pulse-rate, or pronounced psychic excitation; preliminary medical treatment is then required.

Medical treatment, from our ignorance of the etiology and pathology, is, according to Dr Preble, in a state of chaos. It may be directed towards the correction of the supposed underlying neurosis or towards the counteraction of the individual symptoms resulting from the hyperthyroidism. His creed is apparently that of Moebius: "Anything is of value, provided only that the patient is led to think it is." He mentions many different means that may be employed, but despite the discouraging review, thinks that the majority of cases should be treated by medical methods, since the natural evolution of the disease is towards recovery. *Per contra*, "surgical measures should be sought when, in spite of rest, proper nourishment and hygiene, and an intelligent effort at correcting symptoms, the patient steadily gets worse, and operation should be too early rather than too late."

Kocher's paper confirms Barker's stress laid on the need for care in the choice of a surgeon; but in the hands of a careful surgeon the results can only be said to be little short of marvellous. Before operating, great attention must be paid to the strength of the heart, the amount of thyroid intoxication, and the degree of lymphocytosis, relative or absolute, the increase being a fair criterion of the gravity of the case. The technique of the operation is not considered in detail in this paper. He considers an operation should be done if vascular symptoms have appeared in the gland; but if symptoms are severe, preliminary medical treatment is required. The extent and number of operations must be

considered for each case, the aim being to reduce the gland or its blood supply as near as possible to the normal.

Ligation of more than two arteries at once, or excision of more than half the gland should never be attempted. Operation gives the possibility of an entire cure of the disease—the chances depending not so much on the degree of histological change in the thyroid, but on the duration and extent of secondary changes.

A discussion of these papers by Drs Halstead, Billings, and Mayo follows on Kocher's paper. J. H. HARVEY PIRIE.

PSYCHOTHERAPY OF THE HYSTERICAL DIATHESIS. (**Psycho-**
(498) **thérapie de la diathèse hystérique.**) BERNHEIM (of Nancy), *Journ. de Praticiens*, Oct. 12, 1907, p. 641.

IN this paper Prof. Bernheim insists on the ease and certainty with which hysterical crises and the tendency to have them may be cured by simple suggestion. This may be direct, simply impressing the patient with the idea that the attack will not ensue even though you may be at the time actually provoking it through, for example, pressure on a hysterogenic spot. In some more severe cases the suggestion must be indirect, thus—" that in the course of a few days you will be able to stop these attacks." In severer cases he combines suggestion with what is practically isolation. By such simple means, he says, the power of inhibition may be rapidly and firmly developed in every case. There is nothing actually new in the paper, but it is an interesting exposition of the views of a leader of the Nancy school. He claims that the credit of treatment by suggestion belongs entirely to this school, and that it is quite as efficacious in the waking condition as in that of hypnotic sleep. J. H. HARVEY PIRIE.

A VALUABLE SIGN IN EXOPHTHALMIC GOITRE. A. L. BOSTON,
(499) *New York Med. Journ.*, Aug. 17, 1907, p. 294.

THE writer's sign, which he has observed in 15 cases after exophthalmus has become conspicuous, is elicited by getting the patient to rotate the eye upwards, then to follow a finger rather rapidly downwards. It consists in (*a*) arrest of descent of the upper lid; (*b*) slight spasm, with apparent slipping back; (*c*) further closure of the lid. J. H. HARVEY PIRIE.

RESULTS OF REMOVAL AND TRANSPLANTATION OF
(500) **OVARIES.** F. M. A. MARSHALL and W. A. JOLLY, *Trans. R.S.E.*, Vol. xlv., 1907.

THE paper contains an account of experiments in which the ovaries were removed from the normal position in a number of rats, and were transplanted on to abnormal positions (usually on to the peritoneum) in either the same or in other rats. A large proportion of the grafts were successful; but success was not so frequent in those cases in which the organs were transplanted into other individuals (heteroplastic transplantation). Some of the grafts were found to have survived for several months when the animals were killed. It was found, also, that the transplanted ovaries underwent the same cyclical changes as normal ovaries, ovulating in the breeding season and forming corpora lutea. Furthermore, the experiment showed clearly that the nature of the ovarian influence upon the uterus (and so, probably, upon the whole organism) is chemical rather than nervous, since the successfully transplanted ovaries, while performing their functions, had lost their normal nervous connections. AUTHOR'S ABSTRACT.

THE INFLUENCE OF THE BLOOD-RELATIONSHIP OF PARENTS
(501) **ON THE CHILDREN. (Der Einfluss der Blutsverwandtschaft der Eltern auf den Kinder.)** Prof. Dr. E. FEER. Karger, Berlin, 1907.

THE conclusions arrived at by the author are the following:—

(1) It is not proved that there are peculiar or pernicious consequences depending solely on blood-relationship of the parents.

(2) The peculiarities and diseases of the children of parents who are related by blood can be explained by the known facts of heredity.

(3) The rare predisposition to certain diseases, especially to retinitis pigmentosa and congenital deaf-mutism, is more liable than others to be inherited if the disease is present in the families of both parents. J. D. RANKIN.

CLINICAL REMARKS ON THE FUNCTIONAL ELEMENT IN
(502) **CERTAIN FORMS OF ANOSMIA.** P. M'BRIDE, *Brit. Med. Journ.*, Oct. 12, 1907, p. 945.

THE paper begins by referring to the commonly admitted causes of anosmia. A number of cases are then described which can only

be explained on the assumption that the anosmia was functional and not due to organic causes. It would take up too much space to describe all of them, but the most convincing was that of a gentleman who suffered from loss of taste and smell with intermissions, and in whom there was marked enlargement of the middle turbinateds. Gradual diminution of the structures was effected by operative means, and eventually one was entirely taken away. Even this, however, did not prevent relapses. The author, therefore, felt entitled to conclude that the anosmia was not due to obstruction, while at the same time the intermissions proved that there was no organic change of the nerve endings, nerves, or centres. The inevitable conclusion seemed to be that the periodic losses of smell and taste must be described as functional.

The observations of Zwaardemaker, Reuter, and Zarniko are then quoted. The author's views are summed up in the following paragraph:—

"In the cases I have brought forward, facts have been adduced which seem to me to prove that loss of smell in the presence of nasal obstruction is by no means always due to this cause. If the obstruction be very marked, operative measures will be indicated on general principles. The difficulty in arriving at a decision is pronounced in those cases in which the middle turbinateds are somewhat enlarged and may give rise to the symptom. It might be thought, as indeed I did think until recently, that where the sense of smell returned after measures calculated to diminish the enlargement, we have sufficient evidence to justify any expectation of great benefit to be derived from operative procedure. The cases I have detailed—more particularly the first—however, show that this is not so. At one time it seemed to me that, given a case of anosmia with retention of the power of appreciating the flavours of food and an obstructive condition detected by anterior rhinoscopy which might interfere with the entrance of odorous particles, we were justified in concluding that the removal of the obstruction would benefit or even restore the sense of smell. It appeared probable that the preservation of the power of tasting in the above sense indicated a healthy condition of the olfactory nerve. An analysis of my cases, however, negatives this view."

AUTHOR'S ABSTRACT.

PSYCHIATRY.

ON THE SIGNIFICATION OF PHYSICAL ANOMALIES IN RE-
(503) **LATION TO THE ETIOLOGY AND PATHOGENESIS OF DEMENTIA PRÆCOX. (Sul significato delle anomalie fisiche in rapporto all' etiologia e patogenesi della demenza precoce.)** G. SAIZ (of Brescia), *Riv. sperim. di Fren.*, Vol. xxxiii., Fasc. 2, 3.

WITHOUT prejudice to the theory that some form of auto-intoxication is an important factor in the pathogenesis of dementia præcox, the author insists on the fact that this mental disorder develops on a degenerative or hereditary basis. After a brief review of the opinions held by those who have written on the subject, he gives the results of a careful examination of 50 cases of dementia præcox and of 50 attendants with regard to anatomical stigmata, and shows that each patient showed 18 anomalies on the average, while that of the attendants was 9·65. No attendant presented more than 17 anomalies, while 74 per cent. of the cases presented 18 or more anomalies. C. MACFIE CAMPBELL.

MENTAL SYMPTOMS PRECEDING ATTACKS OF HEMICRANIA.
(504) **(Sintomi psichici premonitori dell' attacco emicranico.)** G. GUIDI (of Rome), *Riv. sperim. di Fren.*, Vol. xxxiii., Fasc. 2, 3.

THE author does not take up the disputed question of the existence of a special neuralgic or hemicranic psychosis, but reports 6 cases in which hemicranic attacks were preceded by mental symptoms. The hemicranic attacks were typical, and the premonitory mental symptoms came on one or more days before the hemicrania. The patients showed a sudden change of mood, either of the nature of an excitement with talkativeness, busy activity, and jocularity, or of a depression with irritability and seclusiveness.

The author found these premonitory symptoms well marked in 6 out of 38 cases of hemicrania, and less pronounced in 18 cases.

Sufficient attention has not been paid to this prodromal period in the examination of cases. C. MACFIE CAMPBELL.

ON THE PATHOLOGY OF PUBERTY. (Sobre Patologia de la
(505) **Pubertad.)** C. DE ARENAZA y J. P. RAFFO, *Arch. de Psiquiat. y Criminol.*, July-Aug. 1907.

THE medico-legal report of the case of a boy whose mental development was deficient from birth. At puberty he had an

attack of mania. The author discusses sexual psycho-pathology at puberty, pointing out the physical and mental degenerations which accompany perversions of the sexual instinct.

J. D. RANKIN.

CIVIL INCAPACITY OF THE HYSTERICAL. (Incapacidad civil de las Histéricas.) LUCAS AYARRAGARAY y BENITES, *Arch. de Psiquiat. y Criminol.*, July-Aug. 1907.
(506)

THE medico-legal report of the case of a woman who presented throughout her life a series of typical hysterical episodes; on several occasions she made serious attempts at suicide. No attempt is made by the authors to analyse the case from the point of view adopted by Freud and his school, although they ask the question, "In what form, in what circumstances, and at what period of her existence did her mental instability manifest itself?" They do not seek for an original upsetting factor, but consider the view held by Dejerine, that "Hysteria is a hereditary affection par excellence," a sufficient explanation of the case.

J. D. RANKIN.

A CASE OF DISORDERED PERSONALITY. RICHARD DEWEY (of Wanwatosa, Wis.), *Journ. of Abn. Psych.*, Oct.-Nov. 1907.
(507)

A FRAGMENTARY analysis of the case of a woman of twenty-three who had an incomplete alteration of consciousness for sixteen days. During that time she assumed the name of another person, and spoke of her real self as of a mere acquaintance. Her handwriting was absolutely different from her natural writing. She wrote from right to left and from the foot of the paper to the top, inverting all the letters. This state of altered consciousness was "preceded by several months of pseudo-paranoia—apparently an evolution of systematised delusions, which were taken for facts, however, by her associates—probably a hysterical or histrionic fabrication of the patient." From the age of nineteen she had been subject to attacks of migraine and hysteria and had shown signs of erotomania of homo-sexual character. On recovery from the state of altered consciousness she recollected nothing of it. In the last report of her case, two years later, there had been no further impairment of consciousness. J. D. RANKIN.

TREATMENT.

INTRODUCTION OF DRUGS INTO THE ORGANISM BY MEANS
(508) **OF ELECTRICITY. (Sur l'introduction des médicaments dans l'organisms au moyen de l'électricité.)** DOMENICO D'ARMAN, *Arch. d'Élect. Méd.*, oct. 25, 1907, p. 773.

CONCLUSIONS at which the writer has arrived from clinical observation and experiment:—

1. Physiology and clinical observation have proved that various drug substances may be passed into the animal organism by means of electricity.

2. Physiology has demonstrated beyond question that, under some circumstances, this introduction may be carried out in doses such as may produce toxic, and even fatal effects.

3. All clinical observers have not, however, been able to satisfy themselves of the advantages of ionisation in such cases; the same physician may sometimes obtain in cases which are apparently similar, and using the same technique, different therapeutic results.

4. Cases in which beneficial results have been obtained are chiefly those of arthritis and chronic neuralgia.

5. In most of the cases in which ionisation is indicated, we may ask ourselves whether the introduction of the same drugs by other methods (buccal, rectal, sub-cutaneous) might not have been more simple, more economical, and equally efficacious.

6. But when these methods of introduction have been tried in vain, and when galvanisation itself has not given satisfactory results, the use of therapeutic ionisation is clearly indicated.

7. It would be of great service to make numerous clinical experiments on the efficacy of the introduction of drugs by means of electricity, eliminating as much as possible every cause of error arising especially from the impossibility of distinguishing the action of the drug ions from that of the galvanic current.

8. In many cases intensities beyond 25 m.a. are not tolerated by the patient, even when the skin seems to be in normal condition; it is therefore necessary to determine with precision not only all the details of the proper technique, but also the conditions of the part of the skin to which the active electrode is applied.

9. In our present state of knowledge the question must be regarded as one still demanding study.

ALEXANDER BRUCE.

HYDROTHERAPY AND ELECTRICITY IN THE TREATMENT OF
(509) **NEURASTHENIA.** G. L. KERR PRINGLE, M.D., *Edin. Med. Journ.*, Oct. 1907, p. 320.

THE varieties of neurasthenia which are benefited by spa treatment may be divided into three groups, dependent (1) on toxic states; (2) on fatigue, from worries probably dependent on group 1; (3) on shock, probably dependent on a hereditary unstable nervous system.

Group 1 will be assisted by a course of the Harrowgate sulphur waters, which get rid of the constipation and the toxic materials from the blood. If the patient is unable to take the waters, the same result may be obtained by the Plombiere bath. The general condition of the patient is later "toned up" by the various forms of thermo-mechanical baths, by which the functional activity of the vaso-motor fibres is stimulated. In group 2 the tonic waters, such as the "Kissingen" or "chloride of iron," are prescribed, the debilitated heart being improved by the use of the Nauheim or Schwalbach bath. In group 3 the nervous system must be soothed by means of hot sulphur or peat baths, followed later by the Aix or Vichy massage douche.

In combination with the above, "high frequency" and "static" electricity must be looked on as a very useful addition to the armament of the physician, especially where some local manifestation is present, as, for instance, a neuritis which has had a rheumatic or gouty origin. It must not be forgotten that the benefits accruing from the above methods must in part be attributed to a psychic effect, and also to the personal influence of the physician. AUTHOR'S ABSTRACT.

Reviews

DIAGNOSIS OF ORGANIC NERVOUS DISEASES. HERTER AND CLARK. Pp. 676. New York: G. P. Putnam's Sons. Price 12s. 6d.

THIS is simply a revision and extension by Dr Pierce Clark of Professor Herter's work. The scope of the work is sufficiently well known to make a full review unnecessary; in this edition, however, the diagnosis is more confined to "organic" disease, only a chapter being devoted to the distinction from "functional" disease. It must be admitted that the illustrations are much in-

ferior to what we expect in American text-books, and certainly not on the same level as the text; a few are original, but the majority are very old friends, copied from work to work. The motor cortex is still confidently spoken of and figured as on both sides of the fissure of Rolando. For a work whose main value must be for senior students and practitioners who have no neurological clinic to study at, more elaboration of the chapter dealing with methods would be of advantage. Such statements as the following (p. 626) are not very instructive: "By stroking the sole of the foot it is retracted, which demonstrates the plantar reflex." Under Babinski's sign he says the movements of the other toes are to be neglected; the fan sign is not mentioned, only the great toe movement. These are some of the defects in a little manual which should otherwise serve its purpose well. J. H. HARVEY PIRIE.

DER SEHNERV UND DIE NEBENHÖLEN DER NASE. By Professor A. ÓNODI of Budapest. Pp. 69. 33 Illustrations. Vienna and Leipzig: Alfred Hölder, 1907. Price 6 M.

THE relationship of diseases of the eye, and especially of the optic nerve, to suppurative conditions in the nose and its accessory sinuses has lately been attracting a good deal of attention, and this book gives an excellent scientific account of the subject and literature. The title is not sufficiently comprehensive, as affections of the lachrymal apparatus as well as of the brain and its membranes (including the venous sinuses) dependent on suppuration in the nasal accessory sinuses are all dealt with. The illustrations, which are original life-size photographs drawn from the author's large atlas, are in every way excellent, though one cannot help regretting that allusions are so often made to figures in the author's atlas not reproduced in this book: to anyone not possessing the atlas this must be rather irritating, and tends to destroy interest in the present volume. The book is divided into two parts: I. Anatomical relationship of the sinuses to the optic nerve. II. Clinical observations and results of post-mortem examinations in cases of sinus suppuration complicated by orbital or intracranial inflammations.

In the first part of the book, Fig. 3 is a photograph prepared according to an excellent new method from a formalin-hardened specimen in which the bone has been removed, leaving the living membrane of the sinuses in position, and shows the close relationship of the optic nerve to the sphenoidal sinus and internal carotid artery. Fig. 4 shows the relation of the left optic nerve in one case to the left sphenoidal sinus. The author points out the extreme thinness of the wall of the optic canal—in some

cases only 1 mm., and the presence of dehiscences in the bony wall which would cause a diseased mucous membrane to come into direct contact with the orbital periosteum, dura mater, or sheath of the optic nerve.

The results of the anatomical investigations may be stated as follows:—

1. The optic nerve may not be related at all to the sphenoidal sinus or posterior ethmoidal cells—*i.e.* thick bone may intervene.
2. The most posterior ethmoidal cells on both sides may form the median and lower walls of the optic canal.
3. The most posterior ethmoidal cell on one side may be related to the optic nerve on the same side or to that on the opposite side.
4. The sphenoidal sinus on both sides may be closely related to the optic nerves.
5. The sphenoidal sinus on one side may be related to the optic nerve on the same side or to that on the opposite side.
6. The sphenoidal sinus and the most posterior ethmoidal cell on the same side show close relationship to the optic nerve.
7. The most posterior ethmoidal cell forms on both sides the median wall of the optic canal, and the sphenoidal sinus on both sides forms the wall of the sulcus opticus.
8. The most posterior ethmoidal cell on both sides and the sphenoidal sinus on one side are related to the optic nerve.
9. Both sphenoidal sinuses and one posterior ethmoidal cell are related to the optic nerve.
10. The sphenoidal sinus on one side and the most posterior ethmoidal cell on one side are related to the optic nerve.
11. The frontal sinus may be related to the optic nerve.
12. A cell in the superior turbinal may be related to the optic nerve.

The connection of the ethmoidal veins with the ophthalmic veins and those of the dura mater enables a thrombophlebitis due to sinus suppuration to give rise to orbital and intracranial complications without obvious continuity of the inflammatory process.

At the beginning of Part II., which deals with the recorded cases, Onodi states that our knowledge is very incomplete in regard to the disturbances of vision due to lesions of the sinuses. A few observations have been made where infection has occurred by direct continuity or by the venous system, but only in a few cases has the route of infection been worked out, and much has still to be done in regard to the microscopic and bacteriological investigation of the sinuses and of the optic nerve in these cases;

the condition of the ethmoidal veins, central vein of the retina, and venous branches from the diseased sinuses must also be examined.

Although this is true, the connection between optic neuritis and sinus suppuration can no longer be doubted; the author admits that it is true that both diseases may exist together, and yet operation on the sinus may not influence the course of the optic neuritis; and further, though operation on the sinus be refused, the neuritis may heal spontaneously: in many cases of optic neuritis nothing abnormal may be found in the nose, and, in others, sinus suppuration may only come on after the patient has become blind from other causes, so that each case of optic neuritis which is supposed to depend on sinus suppuration must be closely examined.

The review of the literature and the references given show what endless trouble has been taken to collect cases from all sources: it is impossible to give a general impression of the clinical cases quoted, but one or two may serve as illustrations.

Ortmann has recorded one of sphenoidal empyema complicated by extra-dural abscess in the region of the sella turcica, and thrombosis of both cavernous sinuses in which the microscopic examination post-mortem showed diplococci in the mucous membrane and periosteum of the sinus, in the bone, and also in the dura and pia-mater. In a case of Hajck's of ethmoidal empyema there followed meningitis and acute hydrocephalus; the organism present was the streptococcus pyogenes, and infection in this case had been by way of the veins. Ónodi's own cases are not yet ready, and, as they are being worked up with the help of Dr Entz of the Pathological and Anatomical Institute of Budapest, they ought to throw considerable light on the subject. The series of cases are divided into four groups—those relating to the antrum, frontal sinus, ethmoidal cell labyrinth, and sphenoidal sinus respectively: some of the last are very convincing.

Looking at the subject from another point of view, Ónodi gives a series of cases in which the eye and intracranial contents were affected by direct continuity, secondly by the venous connections of the nasal mucosa and sinuses with the orbital and intracranial veins, and thirdly those in which infection probably passed along the lymphatics surrounding the sheaths of the olfactory nerves: this last connection is not yet made certain by microscopic investigation.

Kuhnt is stated to have observed thrombosis of the central vein of the retina three times in cases of empyema of the maxillary antrum, but the connection was not proved.

Finally, Mendel and Lapersonne are quoted to the effect that *half* the cases of one-sided optic neuritis can be recognised to be of nasal origin; Sattler does not agree with this, and Ónodi is on

the side of the latter observer—in fact, he is most anxious not to claim undue importance for the connection between sinus disease and optic neuritis, and to examine most critically all cases in which an apparent connection is made out. We look forward to the publication of the author's further work on this subject.

J. S. FRASER.

TECHNICAL PROGRESS AND MENTAL HEALTH. WITH AN INTRODUCTION: ON THE EDUCATIONAL VALUE OF PSYCHOLOGY. (Technischer Fortschritt und seelische Gesundheit. Mit einem Geleitwort: Vom Bildungswert der Psychologie.) WILLY HELLPACH. Halle: Marhold, 1907. 2 M.

THIS brochure is one of many nowadays appearing in Germany that attempt to correlate psycho-pathology with sociology, and to draw inferences from the study of the former subject that may prove of value in relation to the latter. The Introduction is concerned with a discussion of the question as to whether the subject of practical psychology, as distinct from philosophy, should be introduced into the Technical High Schools, a question the author naturally answers in the affirmative. This has little interest for readers in England, however, as here we have not yet settled whether the subject should be introduced into the Universities.

Broadly speaking, the theme of the brochure is the relation of modern civilisation to mental disease. The author assumes, a little gratuitously, that increase in the one has proceeded hand in hand with increase in the other, and even maintains that certain forms of mental affection have been created in the past fifty years. He is little concerned with the question of insanity, for he attributes its increase to alcohol, syphilis, and artificial feeding of infants (!), and not to any increasing complexity of environment that progress in technical affairs may have brought with it. Modern psychiatry has something different to say on this subject, which, however, the author does not pursue further. On the other hand he lays the responsibility for the increase of all those affections that the Germans group under the name of "Nervosität" directly at the door of modern civilisation, the essential factor being, he considers, the enormous progress in technical machinery. He divides Nervosität for the present purpose into two great groups: that of the middle classes, which he calls "chronic mental over-stimulation," and that of the lower classes, which he typifies as "work hysteria."

Dealing first with the former group, he opens with the astounding statement that we owe all our knowledge of the subject to

Beard, since whose writings nothing essentially new has been produced. Now all scientific knowledge we have of this subject has certainly been acquired independently of and subsequent to Beard's writings, and is due to the labours of such men as Prince, Freud, Parker, Gross, Janet, Sidis, etc. By a curious chance the author has put his finger on one of the essential factors concerned in the increase of neurasthenia—the greater possibilities of intercommunication—but without any inkling as to how it operates. Indeed he seems to think it is largely a matter of worrying about having to catch trains. It really is time that psychologists begin to analyse what they mean by such phrases as "the hustle and bustle of modern life," "hurry and worry," "restless desire for constant change," instead of constantly repeating clerical platitudes that dismay us with their superficiality and self-assured dogmatism.

Fortunately Dr Hellpach shows considerably more understanding of sociology than of psychology, so that his discussion of the second group is well worth reading, perhaps because he acknowledges that the problems here are less "durchsichtig" than the previous ones. He falls into the common error of thinking that, because neuroses in the lower classes at first sight resemble simulation more than those in the middle classes, therefore the element of simulation and exaggeration is in fact very great. This is curious, for he shows a sufficient knowledge of the sociological origin of this error to have enabled him with a little further thought to avoid it. With striking insight he lays emphasis on three all-important matters, two concerned with the origin of neurasthenia in the lower classes, and one with its prevention. The first two are the constant psychical trauma and conflict that result from—(1) the insecurity of livelihood, with uncertainty and suspense concerning the future; and (2) the lack of interest in work and in life that results from a joyless, monotonous occupation, which presents little or no opportunity for personal advancement or reward. With regard to these social problems that beset a state of affairs in which the majority of the population must necessarily be employed on work of this character, Dr Hellpach boldly reaches out to a solution that few sociologists dare to offer, and which most social reformers are opposed to, namely, such an increase in technical knowledge as to render the number of men engaged in the manual occupations referred to negligible or even nil. We commend this section of his brochure to those interested in social matters, but warn them against disappointment at the way in which the psychological questions are handled.

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